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# ournal of ccupational edicine

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The Industrial Medical Association was organized in 1915 as the American Association of Industrial Physicians and Surgeons. Its first annual meeting was held the following year. In 1951 the present name was adopted.

The purposes of the organization as stated in the certificate of incorporation (not-for-profit) are:

"The object of this Association will be to foster the study and discussion of the problems peculiar to industrial medicine and surgery; to develop methods adapted to the conservation of health among workers in the industries; to promote a more general understanding of the purposes of the medical care for employees; and to unite into one organization members of the medical profession specializing in industrial medicine and surgery for their mutual advancement in the practice of their profession."

Presently, the membership includes approximately 4000 physicians in the United States, Canada, Latin America, and other parts of the world. Physicians "who have an interest in Industrial Medicine" may apply for membership; specialization in this field is not essential. Regionally, the Industrial Medical Association has 19 Component Societies in the U.S. The majority of the Association's members carry their memberships through one of the component groups.

The *Journal of Occupational Medicine*, owned and published by the Industrial Medical Association, is sent monthly to each member as one of the advantages of membership.

The annual business meeting and convention is held in the spring. Since 1945, the convention has been held concurrently with the meetings of other organizations having related interests including the American Association of Industrial Nurses, Inc., and the American Association of Industrial Dentists. This combination of meetings is known as the American Industrial Health Conference.

The Occupational Health Institute, Inc., is a non-profit educational organization created by the Industrial Medical Association to promote the cause of health in industry. Since 1951, the Occupational Health Institute has conducted the program of evaluation, approval and certification of Medical Services in Industry, on the basis of standards established by the Standards Committee of the Industrial Medical Association. This program was conducted by the American College of Surgeons for many years until its transfer to the Association.

The Association's headquarters office serves as a focal point for the Association's activities and through its coordination of information regarding industrial health problems encountered throughout the country, it offers invaluable assistance to members on a host of specific subjects.

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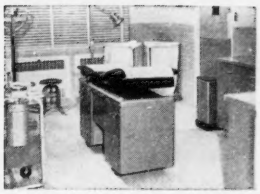

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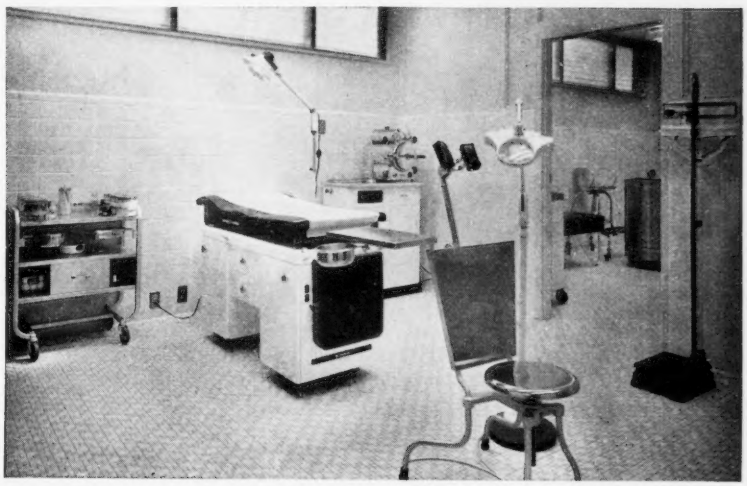

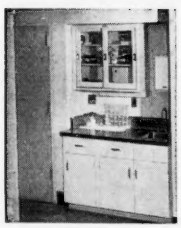

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Through its District Counselors, the Industrial Medical Association is represented regionally throughout the United States and Canada. Each district is served by a Counselor appointed by the President of the Association. The District Counselor reflects the attitude of the membership of his district in his relations with the national organization, and in turn, reports and interprets policies of the Association to the membership and the general public in his district.

An important function of the District Counselor is to serve in a liaison capacity with the Component Society of the Industrial Medical Association in his area. The District Counselor also is concerned with regional trends in industrial medical practice and in legislation bearing upon medical service in industry. He is ready to assist any physician with questions or problems.

In promoting the cause of industrial medicine, many District Counselors work with the industrial health committees of their state medical societies, they participate in teaching programs at hospitals or medical schools, and serve as speakers at meetings of groups that express interest in occupational health. Throughout these activities, the District Counselor attempts to stimulate membership in the Industrial Medical Association by acquainting others with the organization's purposes and facilities.

By keeping in close contact with members in his area, the District Counselor gains a familiarity with their work which enables him to make recommendations to the Association's officers and committees regarding those qualified for various positions or honors. He recommends members for committee work, and for fellowship, and recommends fellows for consideration for elective offices in the Association. He also recommends possible recipients for the awards presented at the annual meeting of the Association—the Knudsen Award, the Health Achievement in Industry Award, the Meritorious Service Award and the Merit in Authorship Award.

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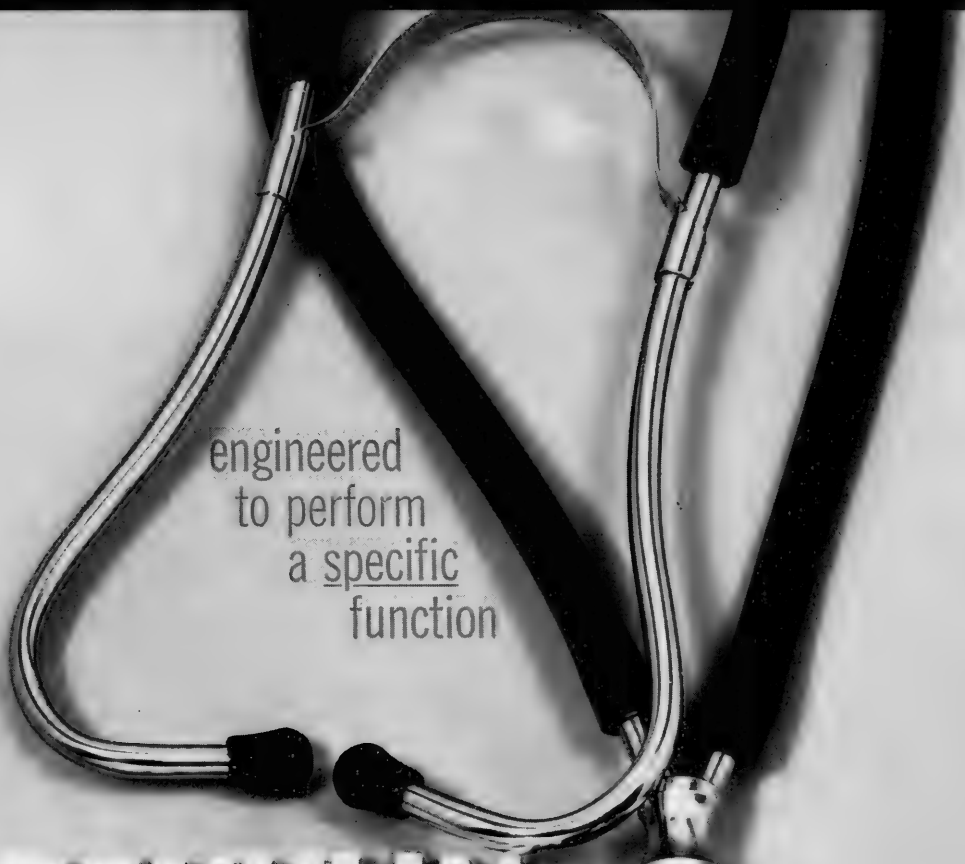


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Journal of Occupational Medicine

# Labor's Interest in Occupational Health and Medical Care

E. Richard Weinerman, M.D., M.P.H., Berkeley, Calif.

WHO CAN speak for Labor—and for whom can Labor speak? Certainly, the labor movement has no one spokesman—least of all me. Many voices represent the organizations of working men and women, with opinions reflecting almost the full range of social and political philosophies in the United States. Yet, in the field of health service, a fairly well-defined position has emerged from the resolutions, writings, and programs of workers and their leaders. I have assumed the task of reviewing and presenting this information based upon my role as medical consultant to various labor health programs, with no pretensions as a union spokesman.

Organized Labor, on the other hand, does claim to represent a larger section of the community than the union membership. The 65,000,000 American workers, together with their families, constitute the great majority of our population.

In a real sense, the 17,000,000 who are organized into local, state, and national union structures are the politically visible and audible representatives of wage earners and consumers in general. Just as working conditions achieved by union effort tend to be standard-setting for the entire labor market, so the social and legislative objectives of Labor reflect the needs of the wider but less articulate general community.

Presented at the Western Industrial Health Conference, San Francisco, Oct. 8, 1960.

## Labor's Interest in Health Service

It is, perhaps, in the very area of health and medical care that the trade union movement can claim with most justification to express the interests of all working people and their families. One bona fide Labor spokesman, Nelson Cruikshank,[1] Director of the AFL-CIO Social Security Department, has put it this way:

> Interest in medical care is interest in the prolongation of life itself for the individual and for his family and his friends. That interest is universal. And, of course, it is an interest not only in the prolongation of life but in improvement in the quality of life so inseparable from the concept of health as a positive entity, not just the absence of disease.
>
> Organized labor's interest in medical care takes on added significance because it represents the largest single body of consumers or recipients of medical care who are in a position to do anything about it. The members of labor unions in this country, . . . organized into effective collective bargaining agencies, have demonstrated that they are in a position to do something about medical care—halting and stumbling and even misguided as their efforts in some instances have been.

In an economic sense, too, Labor has a direct stake in health conservation and in the provision of good medical service. The worker has only one basic resource—one bulwark against family insecurity. This, of course, is his ability to work and to earn, which, in turn, rests upon his physical and mental well-being. Addi-

tionally, it is largely his money, directly or indirectly, which finances the various programs of health service. Labor considers medical care to be as primary an area of concern and social action as do we in the professional ranks of health personnel.

*Labor's Contributions to Health.*—The contributions of the trade union movement to the public health have been considerable. Especially obvious to occupational health experts must be the relevancy of such achievements in protection of the work environment as the eight-hour day, restrictions on child labor, safety measures, and rest provisions. The labor movement claims primary credit for the establishment of workers' protective legislation, minimum wage guarantees, paid vacations, control over women's working status, and the social security programs. Local unions have supported efforts for better public schools, playgrounds, and housing developments in their own communities. Such social and environmental safeguards of health represent a direct contribution to the well-being of all our people.

In addition, the labor forces have been vigorous advocates of legislative proposals and public programs for the extension of health services to the full population. Maternal and child health programs, medical research, hospital construction, care of the aged (as examples) have all benefited from the political support of this most effectively organized of consumer groups.

Further, the labor movement provides the primary support for the voluntary health insurance system in our country. It is not generally appreciated that over 75% of the enrollment of all voluntary medical plans are members of union or other employee groups and their family dependents.[2] Without the tremendous impetus of the recent trend toward health and welfare plans negotiated under collective bargaining, the voluntary plans would not be reaching so extensive a proportion of the population as they now do. There can be no denial of Labor's obvious concern with current issues of cost and services in the medical care field.

Finally, the unions have made one other fairly unique contribution to the American health scene—the labor health center. In a few demonstration areas, largely in the eastern industrial cities, individual unions have expressed their disappointment with limited cash indemnity benefits as purchased from someone else's insurance plan, and have established group practice medical service centers under their own auspices. Some 60 such facilities—ranging from old-line, limited clinics, such as those of the International Ladies Garment Workers Union, to modern, comprehensive service facilities, such as the St. Louis Labor Health Center—are now in operation, adding one further element of choice to the array of medical plans competing for those medical consumers who seek the most and the best for their health dollars.[3]

*Changing Nature of Medical Care*—Meanwhile, the science and the technology of medicine are constantly changing, calling for appropriate adjustments in the methods of providing health care. The economics of medicine are decisively altered by changing patterns of disease, by changes in the composition of our population, and by changes in the mechanisms of paying for personal medical care. At the same time, advances in methods of diagnosis and treatment, new preventive and rehabilitative techniques, emergence of the concept of the medical team (as in cardiac surgery, for example)—all demand the consideration of new forms to reflect the new content.

The more perceptive of Labor's leaders, it must be recognized, are well aware of the dynamics of science and of society. Their call is for health services for their people which reflect the new needs and the new forms. Unsuccessful in their general support for a national health program in the 1930's and 40's, the labor movement now seeks to strengthen the health security of its members on all other available fronts: public programs, voluntary insurance plans, union health centers, demonstrations of new methods of organization and service. On what grounds can we, providers and experimenters in health service, find fault with this motivation or fail to comprehend these concerns?

*Common Interest of Labor and Management in Health Protection*—In many ways, the health area represents one common meeting ground for both Management and Labor, and this understanding increasingly characterizes the industrial health and welfare field. Both worker and manager have a high stake in good health, in consistent performance, in low absenteeism, in family security, in efficient expenditure of medical dollars, in health services of highest quality. Once the contest at the collective bargaining table has been resolved, the parallelism of interest in good medical care is (or should be) jointly recognized.

Unfortunately, however, this common interest occasionally conflicts with the concepts of an older day in industrial medicine. Fixed ideas about the separation of prevention and therapy, about solo versus group practice, about salary and fee methods of reimbursement, about occu-

pational and nonoccupational disorders do not always coincide with the realities of either science or society in the 1960's.

Management, as well as Labor, claims the right to criticize professional dogma when it appears to conflict with other facts of life. The 1956 Lake Logan Conference on Occupational Health, attended by leading industrialists and industrial physicians, was reported by a medical spokesman in the journal of *Industrial Medicine and Surgery* in the following thought-provoking manner:[4]

> It was disturbing to many physicians at Lake Logan to discover that in the minds of some of the industrialists present, the belief [exists] that whatever is good for the workers' health should be put in operation regardless of the restrictions that attend the activities of the industrial physician as part of the medical profession. The industrialists contended that, if care of the worker's family as an aspect of industrial medicine was good for the industrial worker, then this should be brought about and without regard for the industrial physician's oft-stated position—that with rare exception (usually responsive to geographical situations) the care of the workers' families belongs in the area of the practicing physician. . . . *The attitude encountered was that if medical ethics interfered with the total good in connection with the worker's health, then something must be wrong with medical ethics.* (Italics mine.)

## Labor's Interest in Occupational Health

It is against this background of understanding of Labor's interests in health and medical care that we can profitably review its current role in occupational health, on the one hand, and general medical care, on the other.

Perhaps the initial impetus to the trade union movement was the common need and desire among workers in a given establishment to improve their working conditions. From the earliest days of efforts to abolish the sweatshops to the current era of noise abatement and radiation monitoring, unions have concerned themselves with health and safety on the job. Shop steward activities, grievance proceedings, work committees, and the like have consistently emphasized the health aspects of working conditions.

*Collective Bargaining for Health and Safety Provisions on the Job*—Surprisingly, the collective bargaining mechanism has *not* been widely utilized for the establishment of occupational health safeguards, although important agreements have been reached at the negotiation table. As early as 1911, after the famous New York sweatshop strikes, employers and employees jointly established a system of inspection for sanitation and other health conditions. Yet by 1942 a U.S. Department of Labor analysis of 7,000 union contracts showed that only about one-third contained any provision for health and safety on the job.[5] Abrams and I reported in 1951 on representative agreements in California union contracts, including the Painters Union provision for brush washing methods in the prevention of dermatitis, the Mine, Mill & Smelter Workers Union specifications for periodic testing of lead absorption, and the Pacific Coast Marine Safety Code worked out by the longshoremen.[6] More recent contractual agreements concerning specific safety provisions have been developed by the Metal Trades Union and the International Brotherhood of Electrical Workers involving construction in the San Francisco area.[7] The International Chemical Workers Union, long concerned with the problem of diatomaceous earth in the Lompoc plants, have entered into agreements with their employers concerning dust control, periodic chest x-rays, and other preventive measures.[8] A. J. Hayes, President of the International Association of Machinists, speaking before the National Safety Council in 1957, reflects the general attitude of Labor in this field[9]:

> Many unions are thinking in terms of collective bargaining clauses which would require the employer to adopt the safety standards set by the American Standards Association as the minimum acceptable in the shop. . . . Although our efforts will directly benefit only the seventeen million organized workers, perhaps they will release new pressures that will eventually result in improved legislation benefiting all workers.

*In-Plant Health Services*—Labor shares with occupational and public health agencies the conviction that in-plant health and medical services need strengthening and extension. It is recognized that some 500,000,000 days of work are lost each year as a result of illness; that the annual wage loss is over $4,000,000,000; and that at least 10% of this toll is taken by occupational illness and injury.[9] Union spokesmen do seem aware that entirely new problems are arising with automation, use of nuclear energy devices, changing chemical processes, electronics, atmospheric pollution, and new extremes of temperature and pressure. The lack of adequate morbidity recording and the gaps in knowledge of etiology and control are also understood by those responsible for health aspects of trade union activity. But the level of interest and the degree of action on the part of unions is by no means as great for occupational health as it seems to be for general medical care.

Special concern is expressed over the inadequacy of occupational health services in small

plants. Eighty percent of the United States labor force are in establishments of fewer than 500 employees, with fewer than 1 in 20 able to provide adequate health and safety services.[10] A recent California survey found in-plant health programs in only 500 of 300,000 work establishments studied, and reported no such provisions for employees of one of the largest employers of all—the State itself.[11] Demonstrations of the practicability of cooperative plans serving numerous small work places in one community, while not now widely applied by management, are generally endorsed by the labor movement.[12]

*Aspects of Workmen's Compensation*—The labor movement considers the extension of workmen's compensation legislation to all of the states to represent one of its proudest social achievements. The twin principles of cash compensation for wage loss and direct medical care of disability reflect the basic philosophy unions now pursue so vigorously in the field of nonoccupational health service.

Nonetheless, important aspects of the workmen's compensation program are under continued criticism by union spokesmen. One relates to the narrow concept of occupational disease reflected in many state codes. (We are proud of the relatively rational interpretation in the California program, although clarification is still in order with respect to second injury funds, occupational causation and aggravation factors, and the like.) Other areas of dissatisfaction among workers stem from limitations on free choice of physician, insurance company influences upon those conducting disability evaluations, problems of quality of medical services, and delays and administrative red tape in the handling of individual cases.[13]

Perhaps the most serious indictment of current workmen's compensation programs is the inadequate provision of rehabilitation services. Only 4 states support rehabilitation centers; only 5 contribute to rehabilitation services; only 15 provide some maintenance allowances during such corrective care.[14] Jerome Pollack, of the United Automobile Workers, has presented data indicating that, of an estimated 2,000,000 persons sustaining work injuries per year, some 200,000 need rehabilitation services—but fewer than 4,000 of these actually received such care (under workmen's compensation) in one recent year.[13] Many of the problems of "compensationitis," prolonged disability, evaluation bias—to say nothing of the human and economic losses involved—could be avoided if this essential "third phase of medical care" (in Howard Rusk's words) were applied to work-connected disabilities.

**Labor's Interest in General Medical Care**

In most of the areas affecting the security of workers, the efforts of organized Labor have led to the enactment of basic protective legislation. Thus, systems of unemployment insurance, workmen's compensation, disability compensation, old-age and survivor benefits, and the like have established a floor of economic support upon which individual unions could attempt to erect further and special safeguards. In the field of medical care—considered by Labor to represent an equally fundamental need of workers and their families—the bid for such security was lost with the defeat of legislative proposals for a national health insurance program during the prewar and postwar years. Accordingly, unions have felt it necessary to utilize the familiar arena of the collective bargaining process in the effort to secure the basic medical protection needed by their members. In the last 15 years or so, this phenomenon of group health insurance arranged through the place of employment has assumed explosive proportions. This development deserves careful study by those concerned with the occupational health problems of workers. Goldwater actually includes such union health and welfare programs in his definition of the scope of industrial medicine.[15]

Management and Labor have indeed long had "common as well as separate interests in the health and well-being of employees and their families."[16] Employee-benefit plans offered by Management and workers' mutual benefit societies date from our earliest industrial history. Yet, the major development of personal health service plans through group prepayment at the place of work has taken place in the post-World War II era. In 1930 only 2% of the labor force were so covered, while recent figures indicate that 70% are now included.[2, 16]

A number of specific events provided the initial impetus between 1945 and 1950. The wartime freeze and excess profits tax laws gave both unions and employers an economic interest in payroll-deduction welfare programs. A United States Circuit Court decision upholding the validity of collective bargaining for health and welfare plans and the pioneer steps taken in this direction by the coal mine, automobile, and basic steel unions were important stimulating factors. The 1949 regulations of the Wage Stabilization Board extended these activities, and subsequent provisions of the Taft-Hartley Labor Law specified the joint responsibilities of unions and Management for the administration of the funds provided in the bargaining agreements.

Now almost every major industry in the

country is involved with employee health and welfare programs, an increasing proportion of which are negotiated—along with wages and working conditions—at the collective bargaining table. Currently, some 56,000,000 workers plus 84,000,000 dependents have some health insurance coverage through their place of employment. Of this over-all number, more than 16,000,000 workers and 25,000,000 of their family members, or 30% of all covered employees, are in plans specifically established under collective bargaining agreements. About $1,400,000,000 was collected in 1959 in such union-management welfare funds. It bears repeating that all employee-benefit arrangements provide four-fifths of all voluntary health insurance membership in the country today.[17]

*Profile of the Average Union Health and Welfare Program*—Typically, the negotiated union health and welfare agreement specifies a sum of money to be contributed by either the employer or the employees or both, based upon work hours or production units; the funds are managed by joint union-management trustees; group insurance policies are purchased to provide small amounts of life insurance, special disability compensation, and specified hospitalization, surgical, and (less commonly) medical benefits. A recent parallel development has been that of negotiated pension plans, similarly administered.

For the nation as a whole, about 35% of the funds are contributed solely by employers, although the proportion in California approaches 90%. Most of the policies are purchased from the larger commercial insurance companies and the Blue Cross-Blue Shield plans, reflecting their dominance of the general health insurance scene. Nationally, the labor enrollment is fairly evenly split between the commercial carriers and the "Blue" plans, but in California almost 90% are enrolled by the insurance companies.[17, 18]

In those relatively rare communities where full medical benefits are offered by prepaid group practice centers (such as Kaiser-Permanente on the West Coast or the Health Insurance Plan of Greater New York) welfare funds are used for group membership on a direct service basis. A smaller, but potentially even more significant, proportion of union members are provided with medical care through labor health centers of "their own," established specifically for their service in about 60 labor health centers across the country—none, interestingly enough, on the west coast.[19]

Management has, generally, been content to negotiate the funds and leave the process of welfare plan operation to the union representatives. Inexperienced and essentially untrained in the complex matters of insurance and medical care, these union leaders have tended to take the paths of least resistance—the commercial group insurance policies—and to turn details of administration over to the carriers or to insurance-oriented technicians.[1] As a result, the general pattern of cash-indemnity compensation essentially for hospital and surgical expenses has dominated the health insurance aspects of the programs. In the past decade of experience with such plans, Labor has become increasingly aware that this umbrella—while essential for the sudden, catastrophic downpour—does little to meet the worker's basic needs for early and continuous family health supervision, for preventive medicine, for careful diagnostic work-ups and consultations, for medical as distinct from surgical care in or out of the hospital, for management of long-term illness, and for rehabilitation.[20, 21] Yet, for an aging population, increasingly subject to long-term medical disability, these become the truly essential health service requirements.

Moreover, the concern of organized Labor for the nonunion worker and the community in general has led to a reevaluation of the holes in the current health insurance umbrellas: the problems of those changing jobs, the unemployed, the small shop or self-employed, the migrants, the farm workers, the indigent, and the retired. A recent survey by the California Department of Public Health has indicated that no more than 57% of individuals discharged from hospitals in this highly unionized State have any sort of health insurance.[22]

Thus the present paradox: a highly "successful" activity of the trade union movement, providing prepaid medical care through its own direct bargaining efforts to most of its membership, leading now—after 10 or 15 years of experience—to expressions of dissatisfaction with the mounting costs, the limited benefits, the restrictions on preventive and rehabilitative medicine, the tying of membership to the specific job, the lack of professional quality controls, and the exclusion of other significant community groups.

*Current Trends in Union Medical Care Plans*—The dual problem of ever-rising insurance costs and constant membership pressure for more extensive benefits has led to a number of recent trends which are causing concern among both union and management people. Caught in the economic squeeze, both commercial carriers and Blue Cross-Blue Shield plans are tending more toward specified cash indemnity and away from direct service benefits. Group experience rating (with high premium

penalties for those workers most in need of medical care) replaces the original concept of community-wide premium rates. Co-insurance "barrier" provisions further limit the predictable coverage desired by most workers. Demand for better coverage of serious illness costs has led to a recent sharp rise in the use of "major medical" benefits, now affecting some 16% of covered workers.[2]

Rising costs require recurring collective bargaining efforts to secure more welfare funds—simply in order to support the level of benefits already in effect. In addition, employers and unions have wondered about the logic of negotiating payroll deductions to pay for such items as brokers' commissions and insurance company profit retentions. Thus, the increasing interest in self-insured plans in the larger industries.

In reaction to all of these and other problems, voices of Labor are being raised more vigorously in favor of the use of the direct health services of group practice—medical centers—either those available to the general community or those established specifically as labor health centers.[24] The most extensive such program is that of the hospital and clinic network established by the United Mine Workers Welfare Fund. The United Automobile Workers are supporting the new Community Health Association—a nonprofit group practice plan recently opened in Detroit. Reports from the United Steel Workers Union indicate strong interest in a change from commercial insurance to a nonprofit health center approach.[25] Affiliation of local union groups with existing cooperative health centers, such as those in Washington, D.C., and Seattle, Wash., also represents something of a new trend. This desire for medical care rather than cash indemnity is quite clear, for example, in the recent multiple choice provisions of the new Federal Employees Health Insurance Program, where most workers are choosing direct service, full benefit programs—despite higher premium costs—over the partial indemnity offerings of the insurance companies.[26]

Another reflection of the inadequacy of present health plans for workers is the renewed interest of Labor in the social insurance approach. The latest policy statement of the California Labor Federation includes the following in its section on Social Security[27]:

> To organized labor, the development of the voluntary plans did mark an improvement over the practically total vacuum which existed previously. Experience has demonstrated, however, that while in many instances they do meet the needs for hospital and surgical care, they are grossly deficient in respect to meeting the comprehensive needs of the average family and providing incentives for preventive medicine or the promotion of health care.
>
> . . . . .
>
> California labor, recognizing the basic shortcomings of voluntary medical care programs, reaffirms its support nationally for comprehensive prepaid medical care legislation, and dedicates itself on the state level to revitalizing the drive launched under former Governor Warren for a state health care program.

The implications are clear: Although the majority of union members are still enrolled in fee-for-service, cash-indemnity prepayment plans, the dissatisfactions are real. Widespread reappraisal of legislative and health center alternatives is underway.

*Problems of Coordination of Health Service for the Worker*—Heavy Labor criticism regarding the over-all status of health service for workers is directed at the present rigid compartmentalization into in-plant preventive programs, medical care under workmen's compensation for occupational disorders, and private medical care arrangements or separate medical prepayment plans for general and family health service. (Franz Goldman calls this "hardening of the categories.") Workers share the apparent concern of Management (as reported above in the Lake Logan Conference) for the artificial and costly separation of health services needed by the individual and his family. We in the health professions should be the first to appreciate the intimate linkage of home and work environments; the multiple nature of disease etiology; the diversity of aggravating factors in disability; the complex relationships of mental health, nutrition, fatigue, preexisting disease, personal sensitivity, heredity, and family welfare status to so-called occupational illness.

Medical economists report increasing confusion and buck-passing between the industrial medical program on the one side and the mushrooming health and welfare insurance plans on the other. The precise boundary marks established by long tradition among plant physicians, compensation insurance company doctors, family private practitioners and group health center medical teams no longer make much sense to the average worker.

Enough examples exist to demonstrate some alternative approaches. The Kaiser center in Fontana, Calif., provides an integrated service for both occupational and general disabilities, with the obvious advantages for continuity, unit records, and coordinated care. The Hotel Trades Council Health Center, in New York City, provides occupational health examinations

by the same medical group which provides the general medical care. Industrial medical service programs at Endicott-Johnson, in Pennsylvania, Tacoma Smelter, in Washington, and Consolidated Edison, in New York, are further demonstrations of established value. Dr. William Sawyer, medical consultant to the International Machinists Union and others have reported on the several occupational health services which could be rendered by the labor health centers now set up to provide general family medical care.[27]

## Summary

Despite the absence of any single spokesman or inclusive statement on Labor's position regarding health and medical care, major trends and policies can be determined from the many available reports, resolutions, and conferences.

The contributions of Labor to the public health must be recognized as considerable, involving support of workers' protective legislation, improved working conditions, community health services, and development of large-scale union health and welfare programs.

In the field of occupational health, Labor expresses interest (not always actively) in the extension of in-plant preventive and protective services, with special emphasis on cooperative programs among small work establishments. Much attention is given to the need for further improvements in workmen's compensation programs—particularly broadening the definition of occupational disability, raising the quality of medical care of industrial cases and including effective rehabilitation services. The collective bargaining process is occasionally used for the achievement of gains in health and safety provisions on the job.

In the area of nonoccupational or general medical care, Labor's policies and programs are more clearly articulated. Many years of accumulated experience with negotiated health and welfare funds has produced a growing sense of dissatisfaction with the limitations of voluntary, cash-indemnity prepayment plans. Limited benefits, rising costs, lack of quality controls, absence of preventive and long-term care, all have led to increasing interest in the alternative opportunities for broader scope of service and closer control of standards possible under nonprofit group practice medical care plans, including labor health centers operated by unions directly.

The labor movement accepts responsibility for the welfare of unemployed, retired, and migratory workers in the general community; hence the reemphasis upon the role of government to assist in providing needed services which seem to be beyond the capacity of voluntary prepayment plans. The mechanism of social insurance on a national level is favored in this area.

Finally, Labor hopes to see the coordination of all health service for the individual worker—on or off the job, in or out of the hospital, prepaid or compensable, preventive or therapeutic. These objectives would seem to be wholly compatible with those of Management, Medicine, and the general public.

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It is expected that the selection of Fellows will be made on or before February 1, 1962. Address all inquiries to: A.E.C. Fellowships in Industrial Medicine, Atomic Energy Project, University of Rochester, School of Medicine and Dentistry, Rochester 20, N.Y., Attention: Dr. Henry A. Blair.

# The Role of Drugs in Occupational Medicine

Irving R. Tabershaw, M.D., and Charles P. Giel, M.D., New York, N. Y.

ALMOST any industrial physician, if asked whether drugs are important in the practice of occupational medicine, would answer that they play a negligible role. And, of course, he would be right. We have recently completed a survey which shows that the average plant dispensary's expenditure for drugs *and* supplies is not even 2% of its budget.

This situation contrasts sharply with current trends in all other fields of medical care. For example, in 1958 the total bill for physicians' services in the United States was 5.4 billion dollars.[1] Of this amount, obstetrical and surgical fees were 1.6 billion, leaving 3.8 billion for all the other physicians, who do most of the prescribing. In the same year the nation's bill for drugs (ethical and proprietary) was 3.3 billion dollars. This means that the patient spends nearly a dollar on drugs for every dollar he pays the doctor. It is also worth noting that the total hospital bill was 3.7 billion dollars, only slightly higher than the drug bill. Moreover, drug therapy has reached this level only during the past decade. From 1947 to 1959, for instance, ethical drug sales increased three times as much as the gross national product.[2]

The facts behind these figures are known to all of you. Although the foundation for this trend was laid in the '40's with introduction of chemotherapeutics and antibiotics, its full force became apparent only in the '50's. This was the era of broad-spectrum antibiotics, antihistamines, hypotensive agents, steroids, and tranquilizers. Moreover, introduction of the new categories was accompanied by marked augmentation of older categories. In the past decade over 400 new chemicals were added to the armamentarium and the number of new products, compounded, duplicated, or in new dosage forms, ran into thousands.[2] There is every promise that the pace in the '60's will quicken rather than slacken. The point here is not the number of drugs available, but rather the enormous demand for medical care which this growth reveals, the revolutionary advances in treatment which these agents have provided, and the pressure to use them that their presence exerts on the physician.

Yet, although occupational medicine has also grown and developed tremendously since World War II, it has hardly been touched by this revolution in drugs. Occupational physicians probably use drugs less than any other specialists. They do so for reasons, familiar to all of you, which are basically twofold: (1) They are preoccupied with duties which clearly take priority in the industrial environment, and (2) they have regarded nonoccupational disease as forbidden ground.

Historically, occupational medicine developed to perform health functions of prime interest to management. Since industry was primarily concerned with the injuries and diseases for which it was legally liable, industrial doctors became greatly involved in workmen's compensation, including accident prevention and industrial hygiene. Out of this responsibility pre-placement and periodic programs for workers in hazardous jobs developed. As industry became more interested in absence control and group health insurance the occupational physician helped develop and administer programs in these areas. Preventive medicine has been limited to programs such as immunization and health education, applicable to large groups. The only direct approach to general health on an individual basis has been the periodic examination, which in practice is usually offered to a limited number of employees. In summary, occupational medicine has been properly preoccupied with administration, first aid, proper placement, accident prevention, environmental hygiene, and, to a limited extent, diagnosis and mass prophylaxis.

The burden of these activities has curtailed

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the industrial doctor's ability to carry out therapeutic functions. Even more important in limiting his therapeutic role and his use of drugs is the occupational physician's own interpretation of his function—an interpretation which has been conditioned by that of his professional colleagues and the youth of his specialty. This view is reflected in the statement of the Council on Occupational Health of the American Medical Association that care of nonoccupational disorders should be limited to emergency, first aid, or palliative treatment only until the patient can be turned over to his personal physician.[3] Many physicians in industry hew rigidly to the letter of such policy statements.

The industrial physician's interest in, and use of, drugs in the past has been severely limited but there are indications that certain interrelated developments in occupational medicine, industry, the community and the medical sciences may alter the traditional role of the occupational physician. Among these are:

1. *The professional status of the occupational physician.* He is now accepted by national, state, and local medical societies as a specialist, and his organizations are officially recognized. Several schools of medicine and public health offer graduate training on a high level, and recognized residencies exist. Having achieved this status within the medical framework, occupational medicine can now reevaluate its own objectives and proper functions more independently and objectively.

2. *Increasingly effective environmental control.* While industrial accidents and diseases are far from eradicated, the method of attacking them has been fully developed. Application of control techniques to old hazards, and to the newer hazards which new substances and new processes will bring, is largely a matter of engineering. The medical supervision and support for this continuing program requires relatively little of the occupational physician's time.

3. *Greater demands from management.* The very term "occupational," rather than "industrial," medicine reflects a shift in emphasis from accident and disease hazards of large industry; the development of medical departments in banks, department stores, and large offices verifies management's belief in the value of preventive medicine. Parenthetically, "industrial" has come to mean in our society the organization of all productive effort for profit. For example, even banking is now spoken of as the "banking industry."

Moreover, as our labor force becomes increasingly stable, employers are more concerned with the general health of their employees and eager to promote it actively. This is partly due to the additional premium put on skill and experience by the complex and technical nature of modern enterprise. It is also due to the fact that employers are underwriting ever more of the cost of illness through direct sickness benefits and health insurance plans. In addition, management is more cognizant of, and concerned about, the impact of minor illness on productivity.

Sophisticated executives are aware that the physician cannot and should not function as a policeman, but they do expect him to do more than certify the fact of illness. We may, therefore, expect increasing demands for the prevention of minor as well as major illness and for minimizing the disability from either. Interest in health promotion will come to outweigh concern about compensation in more and more companies. In fact, the liberalization of the workmen's compensation laws has taught executives in companies with these problems the value and need for total health programs.

4. *Increasing orientation of the community around industry.* People increasingly look to the source of their economic base for security. Medical care is one of the important elements in the security the employee seeks, and he looks for it, directly or indirectly, from his employer. It appears that the community and individuals would be perfectly willing to accept either preventive or definitive care directly from industry if it were offered. It is quite possible, with the rising cost of medical care, that occupational physicians will be asked by the employer not only to administer these benefits but to supply some of them directly.

5. *Advancing knowledge in the medical sciences.* The greater knowledge of the causes of disease acquired in the last half century has culminated in the therapeutic advances of the last few decades. Indeed, doctors have become so "cure" oriented that thoughtful teachers have decried neglect of the art of palliation.[4] Not enough attention has been called to prevention of the late effects of recurrent minor disorders, such as allergies, nonspecific bronchitis and varicosities, through early active treatment. In addition, the preventive aspects of the more effective treatment of minor disorders without significant sequelae, such as the common cold, tension headaches, and temporary mild depression, in the sense of minimizing disability, has not been sufficiently stressed.

The practical problem of the immediate future for medicine and society is how to distribute most effectively the results of this mushrooming knowledge to the population. It seems reasonable to predict that the ready access of

the occupational physician to large groups of adults who remain healthy or have only mild disorders will cause him to play an increasingly important part in this effort. In short, we face a subtle but vital change in emphasis in the practice of occupational medicine, a shift from environmental control for the group to preventive medicine for the individual. The demand for this change will continue because it arises from the desires of employees, the needs of management, and the progress of medicine. Occupational medicine now has the status to decide for itself how far and how fast it will go in applying preventive medicine. Where will the boundary be drawn in the "gray area" between preventive and curative medicine? The real practice of occupational medicine and with it the role of drugs depends on this decision.

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We recently conducted a survey of 75 plants and found that the drugs used in the average plant dispensary were scanty and disorganized and followed no rational plan. Most occupational physicians apparently accept the orthodox view and provide symptomatic care only until the employee goes home. The agents available included many complex combinations for which none of the staff knew the rationale, usually under brand labels chosen for reasons which had been forgotten. However, a closer look at some of the drugs in some of the dispensaries showed that many of our colleagues are already thinking along the lines we have outlined. A few examples will show what is now being done. They reflect the interpretation that some industrial physicians place on their roles as therapists.

1. *Oral diuretics.* One physician who stocks them uses them in selected cases of premenstrual edema and dysmenorrhea. If his nurse can verify a cyclic weight gain related to menses, he uses them prior to the usual analgesics and muscle relaxants. He has increased the comfort of several women and lessened the absence of a few.

2. *Appetite suppressants.* One doctor's program includes distribution of caloric charts and individual dietary instruction, counseling on the emotional aspects of eating and regular weighing. He has found drugs essential in encouraging people who have unusual difficulty achieving an initial loss, those who reach a plateau, and a few who tend to abandon their diets during periods of stress.

3. *Anticholesteremic agents.* In the course of periodic examinations a physician has found several men with a familial history of circulatory disease, strongly goal-oriented personalities, and moderate, but definite, elevation of the serum cholesterol on serial determination. None has clinical evidence of atherosclerosis as yet. He advised all of them to reduce their dietary intake of cholesterol. The cholesterol level in some on this regimen fell over periods up to six months. He prescribes an anticholesteremic agent for the others and all but one have significantly reduced their blood cholesterol levels with the drug.

4. *Anti-infectives.* These drugs are very commonly used. One physician prescribes prophylactic sulfonamides for some patients with asymptomatic rheumatic heart disease. Other physicians administer antibiotics at the onset of nose or throat symptoms in patients with a history of rheumatic fever or glomerulonephritis. Still others use antibiotics for otitis media or a purulent sinusitis. They feel that absence from work or delaying treatment until the end of the day is unwarranted in these conditions, which respond so readily to early treatment and yet have serious implications if neglected. Many not only administer an initial priming dose, but supply a full course, because they consider it unwise to start an antibiotic without assurance that a full course will be completed.

5. *Antihistamines.* One physician uses these drugs extensively for mild allergic rhinitis and nonoccupational contact dermatitis. He refers severe cases to a personal physician, but he has patients who refuse to visit their doctors for mild self-limited symptoms whose origin they know. He feels the additional comfort and productivity provided by those brief periods of symptomatic control are well worth the expense. Another physician who follows the same principles requires his employees to pay for the drug.

6. *Hypotensive agents.* Several physicians use the milder drugs, such as phenobarbital and reserpine, in cooperation with the private physician. Pressures are checked by the nurse and dosage regulated by the plant physician. Private doctors have been enthusiastic about this regulation, recognizing its convenience for the patient, and appreciative of the fact that each patient is supplied with a card indicating his current dosage.

These few examples graphically portray the response of some occupational physicians to the newer developments detailed above. They are applying available individual preventive measures and doing more early active treatment of conditions they have a peculiar opportunity to see before disability sets in. Yet it is necessary to draw the line somewhere unless occupational medicine is to undertake complete medical care under the guise of prevention in

the broad sense. Except in emergencies, therapy should be initiated only if it can be followed to successful completion. Consideration of this principle, as well as the doctor, nurse, and laboratory time available, will prohibit active treatment of all but the shortest overt illnesses. Even with these limitations there remain three significant areas in which the occupational physician may use drugs as prevention.

1. Preclinical states which can be favorably influenced before disease is clinically manifest. Dietary adjuncts like appetite suppressants and anticholesteremic agents fall into this category. Some, but not all, the hypotensive agents might be included. We may expect more such agents to become available.

2. Early active and definitive treatment for conditions with a serious potential which can be converted to minor illnesses by prompt and effective treatment. Otitis media, acute bacterial pharyngitis, purulent sinusitis, and many skin infections are examples of conditions which would fall into this category.

3. Minor complaints for which the employee might reasonably be reluctant to consult a physician, but whose nature indicates active treatment for a few days to provide maximum relief, permit full job effectiveness, and, most important, limit insidious progression of an underlying process. Minor flare-ups of allergic rhinitis or contact dermatitis fall into this category. Brief, mild bowel disturbances which are recurrent may also be included if organic pathology has been excluded. Dysmenorrhea and mild disturbances of mood attributable to known temporary environmental stresses probably also belong here.

By now most readers have probably said to themselves, "I treat some of those cases," or "I sometimes use that drug." Of course, but in response to immediate needs, rather than out of an organized approach.

It is our thesis that, having decided which minor conditions our nurses may treat under standing orders and which conditions we will treat as part of our preventive efforts, we review the drugs we use and develop formularies which will provide the most effective, least expensive, and safest agents available for our needs.

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# The Humoral Transport of Beryllium

**Andrew L. Reeves, Ph.D., and Arthur J. Vorwald, M.D., Ph.D., Detroit, Mich.**

EVER SINCE beryllium poisoning became recognized as a definite disease entity[1-3] problems concerning its pathogenesis, pathophysiology, and biochemistry have been the subject of numerous investigations. Those problems assumed added importance, especially after the carcinogenic property of the element was demonstrated in rabbits and rats.[4-6] Distribution studies of beryllium compounds in various organs of exposed animals[7, 8] indicated that the material is apparently capable of being transferred from the site of entry or original deposition to distant regions of the body. Since transport via body fluids is the most obvious, and in many cases the only, way of such transfer, the state of beryllium in body fluids is a problem of considerable interest.

Aldridge, Barnes, and Denz[9] in 1949 published an extensive toxicological study of beryllium, and suggested that a stable complex with plasma proteins is formed when a soluble beryllium salt is injected intravenously. This conclusion seemed to be at variance with the results of Scott, Neuman, and Allen,[10] who observed that only tracer concentrations of beryllium are soluble in body fluids, whereas when greater amounts of the material are injected intravenously, most of the beryllium will be in an insoluble form. Feldman, Havill, and Neuman[11] performed dialysis studies of beryllium added to plasma, sera, and aqueous solutions, and suggested that the transport of this ion is determined principally by orthophosphate and citrate anions. Interaction with protein did not take place except with carrier-free $Be^7$, and in the view of these authors, the results of Aldridge, Barnes, and Denz[9] are best explained by the protective colloid action of serum proteins which kept the beryllium phosphate in colloidal suspension. A similar phenomenon in the case of calcium phosphate was noted by Hopkins, Howard, and Eisenberg[12]; the co-sedimentation of beryllium and phosphate in the ultracentrifugal field was confirmed by Reeves, Smith, and Vorwald.[13]

In the present paper, a more detailed investigation is reported on the state of beryllium in body fluids; on the behavior of beryllium in serum during ultracentrifugation and dialysis; and on the action of beryllium on the rat erythrocyte.

## Materials and Methods

*The Artificial Serum*—A number of artificial media were designed with the purpose of duplicating certain physicochemical characteristics of body fluids. These artificial media, or "artificial sera," were compounded in such a way that their properties as sedimentation media should not differ considerably from that of natural human serum. Since any particles sedimenting in the media may be presumed to be essentially round, the parameters governing sedimentability are expressed in Stoke's formula:

$$V = \frac{2gr^2(p\text{-}p')}{9n}$$

where V is the speed of sedimentation, p the density and r the radius of the suspended phase, p′ the density and n the dynamic viscosity of the suspending medium, and g the gravitational force; all values being expressed in cgs units. Accordingly, at a given temperature and at a given geographical location, the speed of sedimentation is proportional to the difference of densities between the suspended phase and the medium, and inversely proportional to the viscosity of the medium. Therefore, specific gravity and viscosity of the artificial sera had to be properly controlled. Furthermore, the artificial sera should contain no protein, so that they may serve as controls for the study of the postulated combination of beryllium with the plasma proteins. Accordingly, the losses in both density and viscosity caused by absence of protein had to be made up by some chemically inert ingredient.

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For transfusion purposes in humans, where true plasma is not available or not indicated, the use of polyvinylpyrrolidone has been introduced.[14, 15] However, our experiments, designed to obtain a polyvinylpyrrolidone solution satisfying the above-mentioned physical criteria, were unsuccessful, because with increasing concentration the viscosity of the solution increased at a far faster rate than its density, and proper adjustment of any one of these two values caused too great discrepancy in the other. The adjustment seemed to be more successful with glycerol, and it was established that a 20% aqueous solution of glycerol has the same viscosity as human serum. The latter value was stated[16] to be 1.75 times that of distilled water at 25°C.

*Table I—Composition of Isotonic Media**

| Ingredient | S | R | R-L | L-L | T | P-C |
|---|---|---|---|---|---|---|
| NaCl | 9.0 gm | 8.5 gm | 8.5 gm | 8.5 gm | 8.0 gm | 6.4 gm |
| KCl | .... | 250 mg | 420 mg | 420 mg | 200 mg | 366 mg |
| $CaCl_2$ | .... | 300 mg | 250 mg | 250 mg | 200 mg | 160 mg |
| $MgCl_2$ | .... | .... | .... | .... | 100 mg | .... |
| $NaHCO_3$ | .... | .... | 200 mg | 200 mg | 1.0 gm | .... |
| $NaH_2PO_4$ | .... | .... | .... | .... | 50 mg | 12.5 mg |
| $Na_2HPO_4$ | .... | .... | .... | .... | .... | 67.6 mg |
| Glucose | .... | .... | .... | 250 mg | 1.0 gm | .... |

*S = saline; R = Ringer's solution; R-L = Ringer-Locke solution; L-L = Locke-Lewis solution; T = Tyrode's solution; P-C = Pannett-Compton solution. All figures are concentrations per liter.

With respect to the inorganic ingredients, the chief objective in compounding the artificial serum was to provide the ionic environment responsible for the buffer pH and the buffer capacity of natural serum. In Table I, the composition of various isotonic media is shown; the condition mentioned above is satisfied by these to a varying, but in no case quite satisfactory, degree. The solution of Tyrode comes closest to the requirement, inasmuch as it has the proper pH, but lacks sufficient buffering capacity. The latter had, therefore, to be replenished in some way, and measurements with the Beckman glass electrode, shown in Table II, established that by increasing the solution's phosphorus concentration to 9 mg% by means of addition of a 4:1 mixture of $Na_2HPO_4$/$NaH_2PO_4$ ("Sørensen's Salt"), the buffering capacity as measured against addition of 0.1 N HCl was raised to that of natural human serum. Accordingly, the fluid so obtained matches well the buffer pH, buffer capacity, and viscosity of natural serum; the discrepancy in specific gravities is 0.02 unit only, and the discrepancy in phosphorus concentrations is 6 mg%. The chief, and from the standpoint of the experiment the only relevant, differences are the lack of protein and the contents of glycerol in the artificial serum. A comparison of the pertinent data is shown in Table III.

*Table II—Buffering Capacity of Natural and Artificial Sera*

| Medium | $P_i$* mg % | pH After Addition of Vols. 0.1 N HCl 0.0 | 0.1 | 0.2 |
|---|---|---|---|---|
| Natural serum | 3.0 | 8.25 | 6.00 | 5.75 |
| Tyrode solution | 1.3 | 7.85 | 4.40 | 3.50 |
| Tyrode solution + 190 ppm Sørensen salt† | 3.0 | 7.40 | 5.25 | 4.75 |
| Tyrode solution + 520 ppm Sørensen salt | 6.0 | 7.40 | 5.55 | 5.30 |
| Tyrode solution + 850 ppm Sørensen salt | 9.0 | 7.40 | 5.85 | 5.70 |

*$P_i$ = inorganic phosphorus concentration.
†Composition of Sørensen Salt: 1 Mol $NaH_2PO_4$ + 4 Mols $Na_2HPO_4$.

*Table III—Physicochemical Parameters of Natural and Artificial Sera*

| | Natural | Artificial |
|---|---|---|
| Buffer pH | 7.35 | 7.40 |
| Specific gravity | 1.04 | 1.06 |
| Relative viscosity | 1.75 | 1.75 |
| Protein content | 5 gm % | 0 |
| Glycerol content | 0 | 20 gm % |
| Inorganic phosphorus content | 3 mg % | 9 mg % |

*Ultracentrifugation*—Aliquots of a beryllium sulfate solution (0.01, 0.10, and 1.00 ml) containing 10 μMol beryllium per milliliter (1.771 g $BeSO_4 \cdot 4H_2O$ per liter distilled water) were added to 10 ml portions of natural and artificial sera. Incubations were carried out at 37°C for 24 hours, under constant stirring. At the end of this period, the samples were ultracentrifuged for five hours at 110,000 × g. Immediately after ultracentrifugation, the tubes were very carefully removed from the rotor and the layers removed with the aid of a 10 ml syringe, fastened in a vertical position and fitted with a long needle. The needle was immersed into the fluid to not more than 1 mm, and the position of the tube was slowly and evenly raised during withdrawal, so as to maintain the 1 mm overlap between the point of the needle and the fluid meniscus. The layers were defined arbitrarily: the top 2-3 ml. constituted the "top fraction" and the next 4-6 ml the "medium fraction." The remainder of the liquid was decanted; this constituted the

"bottom fraction." The sediment pellets were removed mechanically with a stainless steel spatula, and homogenized in 10-ml portions of distilled water for three minutes in the Waring Blendor. All other fractions were also adjusted to 10.0 ml volume with distilled water. The manipulations on the ultracentrifugates were performed on a vibration-free masonry bench; it was hoped that the applied precautions would minimize any error caused by diffusion of the ingredients after completed centrifugation.

*Dialysis*—One $\mu$Mol of beryllium sulfate was added to 10 ml. aliquots of each the natural and the artificial sera. Incubations were carried out at 37°C for 24 hours under constant magnetic stirring. The resulting suspensions were placed into cellophane dialysis bags, and dialyzed at +1°C by hanging them in 3 liter beakers which were filled with distilled water to two-thirds of their volume, and changed daily for six days. The dialyzates were concentrated to about 50 ml volume on the hot plate prior to analysis.

*Analysis for Beryllium*—Quantitative methods of beryllium analysis were reviewed recently by Reeves.[17] In the present study, two of these methods were used: the naphthazarin method of Underwood and Neuman[18] and the spectrographic method of Smith, Boyle, Fredrick, and Zak.[19] Naphthazarin reagent was prepared according to Toribara and Underwood.[20] The spectrographic procedure was simplified and adapted according to Smith and MacEwen.[21] In the spectrophotometric procedure, the Bausch & Lomb "Spectronic 20" colorimeter was used at 600 m$\mu$; spectrography was performed on a Bausch & Lomb dual grating emission spectrograph* by measurement of the 3130 Å beryllium line in combination with the 2057 Å aluminum internal standard line. The spectrographic plates were developed according to standard photographic technique, and evaluated densitometrically. Details of the techniques have been discussed elsewhere.[17]

*Analysis for Phosphorus*—The classic method of Fiske and Subbarow[22] was used. The assay is based on the development of a blue color when phosphomolybdate, formed by treating the phosphate-containing samples with ammonium molybdate, is reduced with aminonaphtholsulfonic acid.

*Analysis for Protein*—Analyses were carried out according to the microdetermination of Lowry et al.,[23] based on a combined phenol-biuret test originally explored and quantitated by Folin and Ciocalteu.[24] Color development was timed, and readings at 50 m$\mu$ against reagent blank were taken exactly 30 minutes after 5.0 ml "Reagent I" (100 vols. 2% $Na_2CO_3$ in 0.1 N NaOH + 1 vol. 2% aqueous sodium tartrate + 1 vol. 1% $CuSO_4 5H_2O$), and 20 minutes after 0.5 ml "Reagent II" (1 vol. stock solution† + 2 vols. distilled water) were added to a sample volume of 1.0 ml. Protein standards ("Versatol" of the Warner-Chilcott Laboratories, Morris Plains, N. J.) were used routinely with each series of determinations.

*Blood Pigment Spectrophotometry*—Different aliquots of freshly drawn venous rat blood were added to distilled water, or to experimental aqueous solutions of varying composition and osmolarity. Pilot experiments revealed that a ratio of 5.0 $\mu$l blood per milliliter of solution is suitable for the 500-600 m$\mu$ spectral range; for the 400-500 m$\mu$ range, a dilution to 0.5 $\mu$l blood per milliliter of solution was used. The optical densities of the resulting mixtures were recorded in a Beckman "DK-2" spectrophotometer.

## Results

*The Ultracentrifugal Experiment*—Table IV shows the analytical results obtained on the ultracentrifugates; the values are medians of three separate experiments.

The beryllium concentrations indicate the migration of this substance in the ultracentrifugal field in all samples, showing that none of the beryllium added to the sera remained in the ionic form. The bulk (or, in one case, all) of the beryllium sedimented toward the bottom and accumulated in a tightly packed pellet. This suggests that the specific gravity of the sedimenting form is higher than that of the medium (= 1.06). In most samples, however, an accumulation of beryllium in the top fraction is also apparent; the effect is somewhat more pronounced in the artificial serum, and is only slightly enhanced with increasing beryllium concentration.

The phosphorus values show only slight tendency toward sedimentation under normal circumstances, and with beryllium concentration up to $10^{-4}$M, no, or almost no, phosphorus appears in the sediment pellet. At a beryllium concentration of $10^{-3}$M, however, a consider-

*Used in combination with a power source of the National Spectrographic Laboratories, Inc., Cleveland, Ohio.

†Fifty milliliters 85% $H_3PO_4$ and 100.0 ml concentrated HCl were added to 100.0 g $Na_2WO_4.2H_2O$, and 25.0 g $Na_2MoO_4.2H_2O$, dissolved in 700 ml of distilled water. The mixture was heated in an electric mantle for 10 hours under a reflux condenser, and, after cooling, a solution of 150.0 gm $LiSO_4H_2O$ in 50 ml distilled water was added. The solution was subsequently decolorized with 4-5 drops of $Br_2$ and diluted with distilled water to a volume of 1,000 ml. This stock solution is stable if kept in a low-actinic bottle in the dark.

able proportion of phosphorus is removed from the solution by ultracentrifugation. There is no significant increase in the phosphorus content of the top fractions comparable to the increase in beryllium in those fractions.

The values of protein concentration are not changed significantly by the presence of added beryllium; the pattern of distribution is almost the same in the investigated 100-fold range of beryllium concentration, as well as in the total absence of beryllium. Any evidence of a beryllium-protein association is absent, and there is no apparent indication that the protein sedimentation pattern is a function of beryllium concentration.

*The Dialysis Experiment*—Two assays were made each with natural and artificial sera, to which beryllium sulfate to a concentration of $10^{-4}$ Mol/liter had been added. The results, shown in Table V, suggest small, if any, difference in the behavior of natural vs. artificial sera. A small amount of beryllium, averaging about 3% of the total, and amounting to only a few tenths of a microgram, escaped daily from the dialysis bags in a random pattern without a discernible time trend. It seems as if an equilibrium mechanism, continuously replenishing this small proportion of dialyzable beryllium, were operative. This mechanism may simply be the solubility of the inorganic precipitate which formed during incubation. The fact that a similar phenomenon was observed in both the natural and the artificial serum samples thoroughly discourages the view that the nondialyzable form of beryllium in this experiment is a proteinate.

*Beryllium and Anaphylaxis*—Since Sterner and Eisenbud[25] proposed immunologic phenomena in the pathogenesis of berylliosis, several attempts[26, 27] were made to explain some or all symptoms of this syndrome on the basis of allergic sensitization. It has been customary to attribute the antigenic effect in these cases to beryllium per se; but it seems somewhat more likely that the true antigen, if one exists, is proteinaceous in nature. It may represent either a normal body protein denatured or altered by contact with beryllium, or it may be the postulated[9] beryllium-protein complex itself. If the concentration of such antigen should rise above a certain threshold value in the host organism, it might induce the formation of antibodies. Repeated contact of the host with the antigen would then elicit allergic, or in extreme cases, anaphylactoid phenomena. An experimental method utilizing these principles may provide additional data with respect to the question of beryllium-protein interaction,

*Table IV—Ultracentrifugal Analysis of Beryllium-Incubated Sera*

| Level of Added $Be^{++}$ | Medium* | Top | Middle | Bottom | Sediment |
|---|---|---|---|---|---|
| | | μg/ml | | | μg |
| $Be^{++}$ Concentrations | | | | | |
| $10^{-5}$ M | A | 0.1 | 0.05 | 0.05 | 0.2 |
| | N | 0 | 0 | 0 | 0.6 |
| $10^{-4}$ M | A | 0.1 | 0.03 | 0.2 | 7.2 |
| | N | 0 | 0 | 0.1 | 8.8 |
| $10^{-3}$ M | A | 0.3 | 0.05 | 5 | 40 |
| | N | 0.1 | 0.07 | 0.5 | 60 |
| P Concentrations | | | | | |
| 0 | N | 18 | 20 | 22 | 0 |
| $10^{-5}$ M | A | 80 | 90 | 100 | 3 |
| | N | 19 | 19 | 19 | 0 |
| $10^{-4}$ M | A | 72 | 73 | 98 | 2 |
| | N | 14 | 16 | 18 | 0 |
| $10^{-3}$ M | A | 88 | 86 | 108 | 28 |
| | N | 6 | 6 | 16 | 34 |
| Protein Concentrations | | | | | |
| 0 | N | 20 | 23 | 30 | 1.2 |
| $10^{-5}$ M | N | 20 | 30 | 35 | 1.5 |
| $10^{-4}$ M | N | 20 | 27 | 35 | 1.5 |
| $10^{-3}$ M | N | 20 | 25 | 35 | 1.5 |

*A = artificial serum; N = natural serum.

and thus aid in evaluating the problem of beryllium transport in blood.

Proceeding on the basis of these postulates, 10 ml of blood was obtained from an adult male rabbit by cardiac puncture. From the blood, the serum was prepared in the customary way and incubated with 1.00 gm of beryllium oxide at 37°C for 96 hours under constant magnetic stirring. Beryllium oxide was chosen in this instance because the earlier clinical work[27] linked this compound somewhat more definitely to the elicitation of the immunological reaction. Although beryllium oxide is very insoluble in water or aqueous solutions, it would not form, for the same reason, in situ precipitates in tissues, as is the case with soluble beryllium. Therefore, it may represent a form more available to surface interaction with proteins, especially on prolonged contact. With respect to these considerations, the incubation time was made longer than the rou-

*Table V—Dialysis of Beryllium Sulfate-Incubated Sera Against Distilled Water*

| Medium | | $Be^{++}$ contents of Natural Serum | | Artificial Serum | |
|---|---|---|---|---|---|
| Dialyzed | 1st day | 0.20 | 0.55 | 0.51 | 0.55 |
| | 2nd day | 0.78 | 0.20 | 0.27 | 0.22 |
| | 3rd day | 0.13 | 0.32 | 0.19 | 0.31 |
| | 4th day | 0.11 | 0.55 | 0.14 | 0.21 |
| | 5th day | 0.13 | 0.36 | 0.12 | 0.26 |
| | 6th day | 0.16 | 0.38 | 0.19 | 0.38 |
| Remained in bag | | 6.33 | 5.64 | 7.29 | 7.85 |
| Total | | 7.84 | 8.00 | 8.71 | 9.78 |
| Recovery | | 87% | 89% | 97% | 109% |
| % Dialyzed | | 20 | 30 | 16 | 20 |

tine period in the ultracentrifugal and dialysis experiments.

The incubated serum was centrifuged to separate it from excess beryllium oxide, and the supernatant reinjected into the same rabbit intracutaneously, in six 0.5 ml doses, each injection two days apart, on six different locations on the shaven abdominal surface. Untreated serum of a control rabbit was reinjected into this latter rabbit in identical amounts and at identical intervals; a third rabbit received Ringer's solution incubated with beryllium oxide exactly as the test serum. It was expected that if the incubated serum contained an antigen, an allergic sensitization of the test rabbit might be induced, distinct from the reaction produced either by serum alone without contact with beryllium oxide, or by beryllium oxide-incubated Ringer's solution alone, without contact with the serum proteins. Upon examination the sites of injections on the abdominal skin were found to be entirely free of the Arthus-phenomenon or any other kind of tuberculin-type allergic reaction in all cases. A larger (3.0 ml) dose of the incubated serum was then administered intracardially, four days after the last intracutaneous injection, in order to elicit the anaphylactoid reaction. This test, too, was entirely negative.

A subsequent experiment, utilizing another rabbit, was performed in order to obtain a precipitin reaction. The animal was sensitized exactly as described above; an aliquot of its blood was obtained prior to sensitization and incubated with beryllium oxide, identically as in the previous experiment. Serum specimens obtained after the presumptive sensitization were then layered in a test tube on top of aliquots of the incubated serum. No visible rings of precipitins were observed.

The demonstration of a beryllium-protein interaction by detecting the antigenic property of the protein involved was, accordingly, unsuccessful by both methods.

*Beryllium and Hemolysis*—In the course of our investigations, attempts to prepare a beryllium sulfate solution of isotonic concentration remained unsuccessful. It appeared that a beryllium sulfate solution, brought into contact with freshly drawn venous rat blood, causes hemolysis in any concentration; at high Be levels (>0.2 M) it also causes crenation of the erythrocytes. It was observed that the color of the hematocrit changed from red to dark brown if the blood was in contact with beryllium, with concomitant disappearance of the characteristic peaks at 412, 535, and 570 mμ of the hemoglobin spectrum (Fig. 1). The

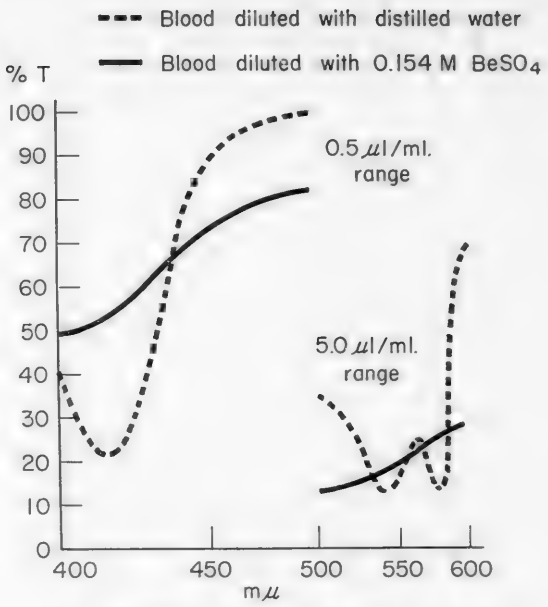


**Fig. 1—Absorption spectra of venous rat blood.**

obvious inference of injury to the erythrocyte membrane, and subsequent acid hematin formation caused by the acidity of the reagent (Fig. 2), was apparently challenged by the surprising observation that if the pH of a physiologic saline solution was adjusted with added hydrochloric acid (final concentration: 0.006 N) to that of iso-osmolar $BeSO_4$ (0.154 M, pH = 2.7), no hemolysis, crenation, or browning of the hematocrit occurred. The effect was thus apparently independent of pH. Tests with magnesium sulfate and lithium chloride showed normal behavior of these reagents as isotonic media, with or without added acid. Accordingly, the impression was obtained that the injurious effect of beryllium on the rat erythrocytes, and its unsuitability to serve as an isotonic medium as observed in these experiments, are chemically specific to this element.

A subsequent trial with solutions of aluminum sulfate, however, showed that the effect is not specific; the phenomena associated with beryllium could be exactly duplicated with aluminum sulfate. The latter solutions have an inherently low pH, without addition of extra acid, due to hydrolysis, similarly to beryllium sulfate. This showed unequivocally that a common physical property of the hydrolyzed salts is responsible for the investigated effect, rather than any chemical specificity. That property is evidently the high buffer reserve associated with continuing hydrolysis. Acidity produced by addition of hydrochloric acid has no such reserve, and is easily overcome by the buffer-

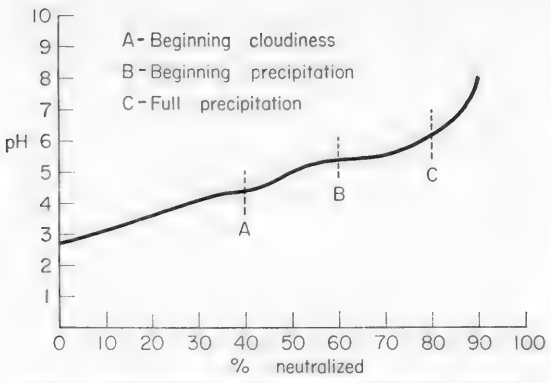


**Fig. 2—Electrometric titration of beryllium sulfate.**

ing capacity of plasma; in case of hydrolyzed salts, the hydrogen ions neutralized by plasma buffers are constantly replenished by a shifting ion equilibrium. The buffering capacity of plasma becomes exhausted and cannot exert a protective effect on the erythrocyte membrane.

The obstacle associated with the preparation of an isotonic beryllium sulfate solution must therefore be considered as fundamental. This property of beryllium is shared by aluminum, but not by sodium, lithium, or magnesium.

## Discussion

The results presented here are generally in agreement with the dialysis studies of Feldman, Havill, and Neuman.[11] These investigators reported that the nondiffusible part of beryllium in serum is present as the orthophosphate; the diffusible part was found to be complexed mainly with citrate anions. The formation of noncolloidal (and nondiffusible) beryllium phosphate is confirmed by our ultracentrifugal experiments. Our results show that this form has a density greater than 1.06, and its precipitation is negligible under a beryllium concentration of $10^{-3}$ Mol/liter. That precipitate, however, is only one fraction of the total serum beryllium.

Under our experimental conditions, the serum beryllium was separable into two distinct fractions; the second fraction, fairly independent in amount of the beryllium concentration, migrated upward in the ultracentrifugal field. Remarkable is the more pronounced tendency of beryllium to manifest this upward migration in the artificial serum samples. However, it is not likely that the absence of protein or the presence of glycerol is responsible for this difference. In order to produce a perfect adjustment of viscosity of the artificial serum, a small discrepancy in specific gravities (1.04 vs. 1.06) had to be tolerated. It would seem therefore that the upward migrating form of beryllium possesses a density of about 1.04 or very slightly less. This could account for the less pronounced, or occasionally absent, flotation effect of beryllium in the natural serum samples.

While the increased beryllium concentration in the bottom and the sediment fractions is matched by a comparable increase in phosphorus concentration, implying the formation of a beryllium phosphate, the identity of the anion associated with the upward-migrating fraction of beryllium is not immediately evident from our results. Our experimental evidence showed only that a phosphate or a proteinate is not involved. Inasmuch as the artificial medium did not contain any organic acids, the formation of citrate in these samples is also excluded. Since beryllium hydroxide has a solubility[28] slightly higher than the orthophosphate,[29] it seems that the hydroxide is the most probable form of upward-migrating beryllium. Its accumulation is especially pronounced at high Be concentrations, where the soluble phosphate of the medium is already exhausted (Table IV).

The protein sedimentation pattern remained essentially unchanged in all samples, irrespective of the fact that the beryllium concentration ranged from 0 to $10^{-3}$ M. This certainly indicates that proteinate formation cannot be a major factor in the transport of beryllium in the blood. The formation of a protein-beryllium complex in very minute concentrations is not necessarily disproved. The importance of pH as a governing parameter should here be emphasized. The studies of Belman[30] showed that definite, albeit reversible, binding of beryllium to bovine serum albumin takes place between pH 3.5 and 5.0. This range, however, is clearly unattainable in vivo. Our studies demonstrated that, if beryllium is allowed to overwhelm the buffer defense of plasma, hemolysis occurs. The high acute toxicity of soluble beryllium[31] may conceivably be connected with this phenomenon. Since this hemolytic property is shared with soluble aluminum, which is otherwise void of known toxic properties, it may be interesting to compare the acute and chronic effects of the two agents on the lungs following inhalation. Such studies are now in progress in our laboratory.[32]

Our immunological evidence, while in itself not conclusive, is consistent with the view that beryllium does not react with serum proteins at physiologic conditions of pH and buffering capacity. While it was recognized that the rabbit may not be so good a choice for the induction of hypersensitivity as the guinea pig, considerations pertaining to the size of the

obtainable blood specimen for incubation required the use of a larger animal. Recently, Scheel[33] reported successful sensitization of rabbits to atmospheric gases attached to egg and milk proteins.

In the solubization of beryllium, the role of complexing anions such as citrate may not be as essential as previously supposed. In our dialysis experiment, comparable amounts of beryllium were diffusible in both natural and artificial sera; the latter contained no organic acids. It seems probable that precipitation of the colloidal forms of beryllium phosphate and beryllium hydroxide is favored when their concentrations are low; these colloidal forms may have a considerably higher solubility that the classical values based on crystalline preparations.[28, 29]

On the basis of these investigations, our concept of the humoral transport of beryllium is as follows:

If soluble beryllium enters the mammalian organism by any parenteral route, immediate precipitation of beryllium orthophosphate takes place in situ. Most of this precipitate remains colloidally dispersed if the beryllium concentration is below about $10^{-3}$ Mol; at that level, the precipitate flocculates with a density of more than 1.06. A minor portion of beryllium (2%-3% at concentrations up to $10^{-3}$ Mol) becomes precipitated as the hydroxide; the amount increases sharply when plasma beryllium attains a concentration high enough to exhaust all plasma phosphate. This precipitate is very light (density about 1.04) and more soluble than the orthophosphate. The colloidal precipitates are in dynamic equilibrium with any noncolloidal deposit formed in situ, as well as with traces of the ionic portion; the "biological half-life" of the element in a certain organ is dependent on the relative amounts of the three forms. In circulating blood or lymph, the colloidal part is probably preponderant. Significant reaction with plasma protein does not occur at biological conditions, and results concerning complexes obtained in vitro at low pH are not applicable in the interpretation of the biologic transport of beryllium.

## Summary

Ultracentrifugal, dialysis, and immunological techniques were utilized to investigate the state of beryllium in body fluids. In all of these experiments, serum specimens were incubated with either beryllium oxide, or beryllium sulfate, at physiologic pH and temperature. In the in vitro study, a properly buffered 20% aqueous glycerol solution served as a protein-free control medium. During an attempt to prepare beryllium sulfate solutions of isotonic concentration, it was learned that such solution cannot be obtained because of the low pH and high buffer reserve of the hydrolyzed salt.

The immunological study, evaluated by the allergic and anaphylactoid reaction as well as by the precipitin reaction, failed to show the development of antigenic property by the beryllium-incubated sera. The results were consistent with the assumption that no significant extracellular beryllium-protein interaction takes place in vivo. The dialysis experiment, likewise, demonstrated that presence or absence of protein in the medium has no effect on the diffusion rate of added beryllium at a concentration level of $10^{-4}$ Mol/liter. The ultracentrifugal experiment, in addition to corroborating these results by means of demonstrating identical sedimentation patterns of protein at widely different beryllium concentrations ($10^{-3}$-$10^{-5}$ Mol/liter), as well as in the total absence of beryllium, also showed that the added beryllium is separable into at least two distinct fractions. Both possessed well-defined migration rates in the ultracentrifugal field, indicating that ionic beryllium in excess of 0.01 $\mu$M/ml was no longer present. A form having a density greater than 1.06 appeared to be the orthophosphate. Another form, having a density of about 1.04, seemed to be the hydroxide. The amount of this latter precipitate was minute as long as total $Be^{++}$ concentration did not exceed $10^{-3}$ Mol. An association of beryllium with the serum proteins was not demonstrated.

These results suggest that precipitation of orthophosphate and hydroxide is the form of existence, and mode of transport, of beryllium in body fluids, including blood, and, presumably, lymph, and the interstitial juices of pulmonary tissue.

## Acknowledgments

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# A Study of Prolonged Absenteeism in Industry

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A THREE-YEAR study of absenteeism at Weirton Steel Company has been concluded by the medical department with some significant results. The study was initiated by the insurance department when the Sick and Accident Insurance Fund became seriously depleted from monthly deficits over a period of several years. The medical department was asked to examine some of the claimants to determine whether they were actually ill and entitled to benefits, to give advice and help to those who were ill to speed their recovery, and to identify factors responsible for the high insurance costs.

The following criteria were used by the Insurance Department in selecting employees to be examined when absent from work due to sickness or injury:

1. A subjective symptom recorded as the "diagnosis."
2. A disability which seemed to be prolonged beyond the usual period for that illness.
3. A claim not submitted within a reasonable time.
4. An employee's absence from his residence.
5. A certification which indicated an extended period of disability prior to the date of first treatment.
6. A certification which did not appear to be absolute and clear cut.
7. A certification which indicated the employee might have returned to work, though he did not do so.
8. A prolonged illness by an employee whose physician was known to be "more than lenient."
9. An illness by an employee who had had prior illness of a troublesome nature.
10. An advisement from the medical department.

## Illnesses Causing Prolonged Absenteeism

Selected by these criteria, 130 persons were interviewed in the Medical Department, almost all by the author personally.* In each case, effort was made to determine the basic disease or problem present. Secondary factors were also identified if possible. Table I depicts the primary diagnoses as so analyzed.

It was determined that psychiatric illness was the principal reason for the absence of 79 persons, 61% of those examined. Emotional disturbance was a paramount cause of illness, and any related organic manifestations were minor in role. The majority of these people were not psychotic, but individuals who meet their problems of adaptation to life in our society poorly and develop multiple somatic symptoms. They will be elaborated on later.

In the psychiatric group I have also included as malingerers 12 employees who were collecting sick benefits although obviously not ill. This number may be considerably larger. In an effort to avoid such a diagnosis, most statements made by the employee were accepted as accurate medical history. It was also recognized that somatic symptoms can develop as a result of unconscious mechanisms in some persons who thereby receive financial benefit (psychodynamically referred to as secondary gain). Those labeled malingerers were not of this type. The remainder of the psychiatric

*Table I—Illnesses Causing Prolonged Absenteeism Among 130 Selected Employees*

| Primary Diagnosis | | Number | Percent |
|---|---|---|---|
| Psychiatric illness | | 79 | 61 |
| Neurosis | 50 | | |
| Malingering | 12 | | |
| Schizophrenia | 6 | | |
| Alcoholism | 3 | | |
| No diagnosis | 3 | | |
| Depression | 2 | | |
| Sociopathy | 2 | | |
| Cerebral deterioration | 1 | | |
| Duodenal ulcer and anxiety | | 5 | 3.7 |
| Pulmonary insufficiency | | 10 | 7.7 |
| Arteriosclerotic heart disease | | 7 | 5.4 |
| Low back Syndrome | | 5 | 3.7 |
| Chronic nephritis | | 2 | 1.5 |
| Single diagnosis | | 22 | 17 |
| Total | | 130 | 100 |

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Dr. Phillips is Assistant Director of Medical Services, Weirton Steel Company.

*During this period, the average regular employment of the Weirton Steel Company was about 13,500 persons.

group is made up mostly of psychotic persons.

Listed next is a group of five persons with duodenal ulcer who are felt to have very strong emotional components to their illnesses, but for completeness are listed separately. They probably belong with the psychiatric group.

The second largest category, which represents a considerable drop numerically from the psychiatric one, is composed of 10 men with pulmonary insufficiency from chronic bronchitis and pulmonary emphysema. It is felt that the finding reflects the importance of this long-neglected disease entity as an increasing cause of disability. Seven persons had arteriosclerotic heart disease, five had various low back syndromes of definite organic nature, and two were ill with chronic glomerulonephritis. All others (22 persons) had separate, single diagnoses as a reason for their prolonged illness.

**Statistical Factors Related to Absenteeism**

The relationship of age, years of employment, and sex to the absenteeism of employees is analyzed in the next tables. Thirty-two (24%) of those interviewed were of the female sex, despite an employment rate of only 3% female employees. One also noted that these female employees are rather young, of very recent employment, and almost exclusively psychiatric problems including malingerers. It must be pointed out here that the female employees as a group are of young age and little seniority. There is a company rule prohibiting continued employment after marriage (unless a hardship case), and some of those investigated and interviewed had attempted to draw sick and accident benefits after they were married, a recognized abuse of the insurance plan.

Among the males (Tables IV and V) the importance of age and length of employment is less readily weighed. It is observed, however, that the number of young men with extended periods of illness is unexpectedly high, and, moreover, that they comprise more of the psychiatric disorders than those in the older age group with more years of employment.

It is concluded from these data that among employees interviewed there was an inappropriate number of females and younger persons with extended absences. Moreover, psychiatric disease was the principal form of illness among them.

**Personality Traits Related to Absenteeism**

The large number of mentally and emotionally ill persons who are responsible for so much of the absenteeism masquerade, as it were, under a variety of organic diagnoses. Frequent among these are peptic ulcer, hemorrhoids, malnutrition, Ménière's syndrome, back strain, chronic bronchitis, hypothyroidism, influenza, and colitis. Neurasthenia, nervous exhaustion, and nervous stomach are a few other "diagnoses" often reported by the medical attendants. But a discerning interview will readily disclose that the prolonged absences of such employees are not really due to any of these diseases. What becomes evident to the interviewer are certain types of psychiatric patterns.

Many of these people are best characterized as inadequate personalities. They are deficient in intellect, and often they are unkempt in appearance, with poor body and dental hygiene.

*Table II—Psychiatric Disease by Age Groups Among Female Employees*

| Age | Number Examined | Psychiatric Disease | Percent of Total |
|---|---|---|---|
| 20-29 | 13 | 11 | 85 |
| 30-39 | 10 | 10 | 100 |
| 40-49 | 7 | 5 | 71 |
| 50-59 | 1 | 1 | |
| 60 + | 1 | 1 | |
| Totals | 32 | 28 | 88 |

*Table III—Psychiatric Disease by Length of Service Among Female Employees*

| Years of Service | Number Examined | Psychiatric Disease | Percent of Total |
|---|---|---|---|
| 1-5 | 16 | 14 | 88 |
| 6-10 | 9 | 9 | 100 |
| 11-15 | 5 | 5 | 100 |
| 16-20 | 1 | | |
| 20 + | 1 | | |
| Total | 32 | | |

*Table IV—Psychiatric Disease by Age Group Among Male Employees*

| Age | Number Examined | Psychiatric Disease | Percent of Total |
|---|---|---|---|
| 20-29 | 14 | 9 | 64 |
| 30-39 | 24 | 17 | 70 |
| 40-49 | 27 | 16 | 59 |
| 50-59 | 22 | 7 | 32 |
| 60 + | 11 | 2 | 18 |
| Total | 98 | 51 | 52 |

*Table V—Psychiatric Disease by Length of Service Among Male Employees*

| Years of Service | Number Examined | Psychiatric Disease | Percent of Total |
|---|---|---|---|
| 1-5 | 12 | 5 | 40 |
| 6-10 | 22 | 16 | 72 |
| 11-15 | 29 | 18 | 62 |
| 16-20 | 6 | 4 | 66 |
| 20 + | 29 | 7 | 24 |
| Total | 98 | 50 | 51 |

They are, in addition, saddled with anxieties and poor ego development. Unable to cope with the problems of day-to-day living, they take refuge in illness, in schizophrenia, in self-pity, and in dependency. Their economic and other troubles are often made worse by their inability to work, but despite this they are unable to "get going." They tend to migrate to the nonmedical healers and to the less skilled among the medical community, avoiding as it were, the means of getting well.

Others have emotional conflicts arising from unhappy and insecure childhoods where a parent was missing through divorce or death. They have a long history of illness back through adolescence and childhood. Statements often made by them are, "Some days I just don't feel good"; "My stomach is killing me"; "They never seem to find out what is the matter with me."

Another type is the sociopath. He can be identified by a history of repeated injuries, both on and off the job, and antisocial acts dating from childhood—fighting, motorcycle riding, vandalism—all arising from impulsive aggressive behavior and self-destructive impulses. Frequently he comes from a broken home, has left school impulsively, may be divorced.

Finally there are the persons who after careful consideration must be challenged as malingerers. They often time their "illness" with a period of contemplated lay off, spring planting or harvest time, or during cold weather for a trip to a warmer climate. Frequently they have a business on the side, or another member of the family is employed.

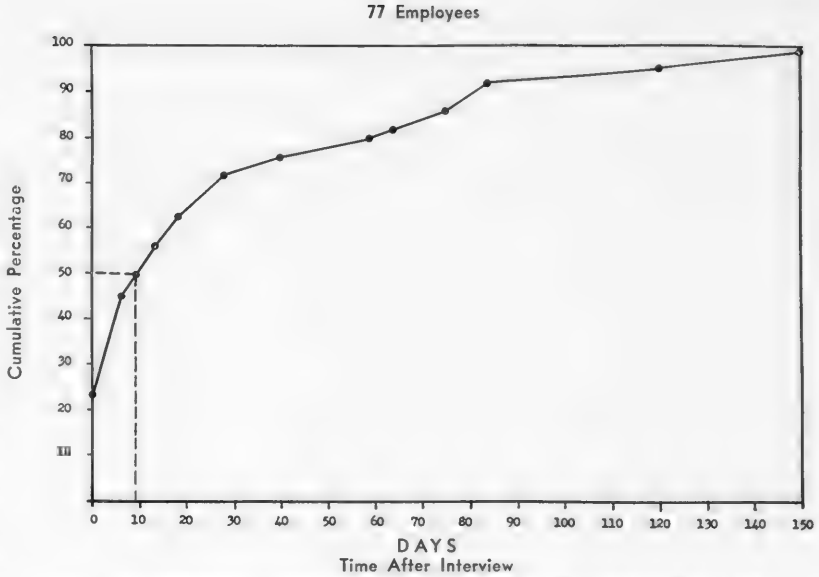


**Fig. 1—Chronological relationship between interview and return to work.**

### Results of Interview and Examination

Table VI shows what happened to this group of persons after they were called in and seen in the medical department.

It may be seen that about 30% of the employees returned to work within one week. This is surprising when one considers that most of them had been absent from work for many weeks to months. Figure 1 depicts graphically the chronologic relationship between the interview and the return to work of the 77 employees who did return. It is shown as cumulative percentage, and the slope of the curve is quite sharp in the period of time closely following the interview; in fact, the 50 percentile is at about 10 days. The connection between the interview and this development seems evident, but it is harder to evaluate for those returning after one week's time had elapsed.

I should like to call attention to the fact that some sort of definitive disposition was made for another 30% of these persons. About 15% were found to have illnesses of such severity that return to work in the future would not be practical, and they were given permanent

*Table VI—Results of Interview and Examination Among 130 Selected Employees*

| Result | Number | Percent |
|---|---|---|
| Returned to Work | | |
| Immediately (within 24 hours) | 18 | 14 |
| Within 1 week | 17 | 13 |
| Within 1-2 weeks | 9 | 7 |
| Within 2-4 weeks | 11 | 8.4 |
| After 4 weeks | 22 | 17 |
| Retired on disability | 19 | 14.6 |
| Released—unsuitable for work here | 19 | 14.6 |
| Voluntary retirement | 1 | 0.7 |
| Still absent, ill | 9 | 7 |
| Status not certain | 2 | 1.5 |
| Denied sick benefits for trip to Arizona | 2 | 1.5 |
| Died* | 1 | 0.7 |
| Totals | 130 | 100 |

*Died of renal failure from glomerulonephritis one month after interview.

and total disability pensions at the termination of their sick and accident benefits. Efforts were made by the medical department and insurance department to aid them in qualifying for social security payments under the provisions amending the law to allow disabled persons to receive payments even though not yet 65 years old. The other 15% were considered to be neither physically nor emotionally suitable for carrying out the types of work available to them in the industry, nor was it considered that treatment and other measures would result in appreciable change in their problem. Accordingly, they were released from employment. Also included in this group are a few persons who were considered well enough to return to work but who did not do so, and a few female employees who were found to be married.

It is felt that the program is of benefit to the employee in the following ways:

1. It helps to evaluate his illness and general health.
2. It encourages him to seek and follow through with treatment if this is not being done.
3. It helps to secure aid for himself or his family through public agencies if this is necessary.
4. It clarifies any problems within the insurance program of the company.
5. It helps to plan any needed work readjustments early.
6. It encourages him to return to work when able to.

**Summary**

The role of a medical department in one aspect of the sick and accident insurance program has been described in which interview and examination of selected individuals was carried out.

It was found that this group was weighted heavily with females, young persons, and recently employed personnel.

Psychiatric and not physical illness was the principal reason for their absence from work.

The program as described unexpectedly proved effective in returning a substantial number of employees to work. It also was instrumental in the definitive disposition of others. It is felt to have been considerable help to the employee himself in several ways.

The over-all effect in reducing insurance costs was considerable.

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# Evaluation of the Whole Man

Jean Spencer Felton, M.D., Los Angeles, Calif.

AT THIS point in time when schismatic developments are creating new and more finite specialties in science, it might appear somewhat quaint to direct attention to the holism of man. As we learn of such disciplines as bionics and molecular biology, and of the deepening of our genetic knowledge through the study of DNA, a suggestion to return to an appraisal of the totality of a patient conceivably could be deemed heterodoxy.

To perform well in a professional undertaking in occupational health, it is necessary to revitalize the concept of the whole man. Any effort, in the medicine of work and industry, to subdivide the employee-patient into spheres of specialty interest will result only in a partially known, partially understood, and, in a sense, prealienated person. Contributions of the social sciences have demonstrated to us the fallacy of intentional or accidental neglect in considering all of the ecologic factors influencing man in his illness. The recording of a clinical history and the completion of a physical examination without appropriate inquiry into the emotional, social, occupational, and spiritual drama going on in the patient's life is comparable to executing a delicate archeologic search with gigantic earth-moving equipment. Not only would significant artifacts be covered but also irrevocably broken during such a blind onslaught.

## Who Is Concerned in the Concept of Holism?

Occupational health is an activity representing the joining of skills of several practitioners, each functioning creditably in his area of industrial hygiene, nursing, medicine, environmental health, safety, or epidemiology. The adequacy of the job done by each of these team members is measured by different criteria. Did the patient get well? Was he motivated into action by the counseling? Did the plant manager approve a new exhaust ventilation system? Were the findings of the statistician followed up for cause-identification? Were available protective devices used in potentially hazardous activities?

Each effort concerns the relationship of one of the health group with a person in the plant, whose role might be that of patient or executive and who contributes to his own welfare or that of others because of the better understanding of him as a person by a physician, a nurse, or a hygienist. In essence, all are concerned with this holistic approach to the worker, and no positive results will accrue in occupational health until its acceptance by the medical team.

## Psychophysiologic Relationships

Medical students for a quarter of a century now have had a good comprehension of "psychosomatic" medicine. Teaching of the behavioral sciences has been introduced in the first year of numerous schools, and the early assignment of a student to a family for which he serves as a counselor has underscored the interplay among those factors other than microorganisms which can produce illness. At our own medical school, teaching conferences in preventive medicine are held with senior students, wherein these elements of impact are reviewed at length in the study of patients whose problems are broader than their physical findings.

In spite of these curricular goings on, we do have many physicians and nurses in industry—full-time, part-time, or on call—who cannot see the patient for the lesion, and who thus miss many of the items of significance which retard the patient's progress. It is not the intention to belabor this point without vivifying it with examples of real-life blood, sweat, and tears. One must emphasize the fact, though, that difficulties are created by patients, too. The mechanism of denial, which is sometimes a patient's only defense against anxiety, precludes even the knowledgeable physician or nurse from painting the full diagnostic canvas.

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Dr. Felton is Professor of Occupational Health, University of California, Los Angeles.

If the patient is motivated inadequately, he might not share crucial etiologic data with the practitioner, and the understanding of the total person will not progress beyond surmise. Deducing a possible etiology from behavioral symptoms, and confronting the patient with the true nature of his problem, is the most delicate of therapeutic tasks.

**Commissions and Omissions**

To paraphrase, one cannot tell the program without the players; hence, here are some who have suited up and have played or have been played badly:

EXAMPLE 1.—A 39-year-old mechanic's helper was viewed by a plant medical department as a potential claimant. On being employed, he had informed his foreman that he could not work at heights. Even with this knowledge, he was assigned to a point high on a ladder below the wing of an aircraft undergoing modification. He fell, striking his head. The patient, in spite of negative findings on examination by several highly qualified physicians, could not work. In a quiet review session with this man, it was learned from his employment application that he was classified 4F during World War II. When asked the reason for this, he stated that he believed this was a result of his inability to read and write. His ego strength was being diluted further by his 9-year-old son who was now surpassing him in literacy. The cue was picked up, and the patient was enrolled in an adult education class at the local high school. He learned to read and write, he returned to work within three days of the interview, filed no claim against the company, and performed well in subsequent weeks.

*Moral.*—Identify the weaknesses as well as the strengths, and use all modalities of therapy.

*Tactic.*—Diversionary.

EXAMPLE 2.—A 46-year-old crate-maker had had an appendectomy four weeks prior to his dispensary visit and had declared himself unable to work because of persistent right-lower-quadrant pain. He, just as the patient in Example #1, had been examined by several surgical specialists and declared physically fit to return to work. The present physician examined him carefully after assuring himself *and* the patient that he understood the symptoms well. The healed incisional scar was slightly tender superficially, but otherwise no significant findings were encountered in the right lower quadrant. Knowledge of his home life was sought, and it was learned that his 10-year-old son was doing poorly in school and that his wife had left him and had been seen with several other men. She had begun legal action to gain possession of their home, his mother-in-law had derogated him in conversations with his friends, and he was, to understate it, depressed. Efforts were made to reach the man's wife for counseling or therapy, through a casework agency and through their minister. This patient returned to work the morning following the initial interview. He was reassured about the operative site, was given supportive heat to the scar for four mornings only, and was seen semimonthly for follow-up reviews of his home situation during the next month.

*Moral.*—Seek other possible etiologic factors when a healed lesion, negative on examination, continues to cause pain.

*Tactic.*—Replacement. Use social casework in place of nonresponsive medical care, but do not eliminate all medical treatment immediately.

EXAMPLE 3.—A 35-year-old carpenter developed pain in the lumbar area with progressive stiffness of the back and lower extremities and inability to walk. Sympathizing, well-wishing co-workers built a special chair in which he could sit and work. Examination findings for protruded intervertebral disk were inconclusive. Consultation with two neurosurgeons and one orthopedic surgeon produced a consensus of insufficient indications for exploratory operation. Material resulting from psychologic testing and interview pointed up the "need" for surgical intervention. With great hesitation, at this point in the patient's downgrade course when walking was almost impossible, the orthopedist agreed to explore and did a laminectomy. The disks seen were normal, and the wound was closed. He had an uneventful recovery and left the hospital within a week, fully ambulatory, and returned to his job at 100% capacity after another seven days. What was the reason as suggested by the consulting psychologist? His mother, when the same age as he, had "back trouble," had no surgery, and was invalided to bed the rest of her life. This the patient did not wish.

*Moral*—Integrate *all* diagnostic information.

*Tactic*—Action, forceful and supportive.

EXAMPLE 4.—A 53-year-old assembler reported that he had struck his leg on the sharp corner of a metal conveyor system. The resulting contusion healed within a few days, but the complaint regarding discomfort at the site remained at high pitch and great intensity. Further, he was adamant in refusing to return to work. On interview, the patient was asked about his job, its components, and his satisfaction with it. The job consisted of his being given a make-up list of items for placement on a tray, which was then sent down the roller-conveyor to the packaging department. His work had been good up to a day or so before his injury. On further search, it was learned that he could not read, a fact which had escaped nearly everyone in the plant. A friend of his at the next point on the line would read his lists for him, and thus he was able to function as an assembler. His friend became ill, and with his reader gone, the patient panicked for fear his foreman would learn of his inability to decipher the lists. His "injury" allowed him to remain off work until his co-worker returned. With this knowledge, and with the cooperation of the foreman, he was reassigned to a satisfying job where no reading skills were required.

*Moral*—Seek occupational factors which might influence the status of health.

*Tactic*—Utilization of skills in place of termination of employment.

This quartet of tales from the factory woods

points up some of the important factors which, if undiscovered, can continue to prolong an illness without the therapist's knowledge of their operation.

As a frequent concomitant, the physician or nurse will blame the patient for being difficult or for failing to recover and soon will develop a dislike for the employee, for he did not behave as did all other patients and get well according to a preunderstood protocol. When the causally related aspects are not known, the professional person in attendance becomes frustrated and, not knowing what to do, labels the patient "a neurotic," or "a chronic," or "a crock," and finds himself utterly unable to treat the person at all adequately. When anger appears, reason leaves, and the relationship can be impaired for all time. The patient, in turn, will go elsewhere for satisfaction, and this could be to an attorney, a union steward, a nonallopathic practitioner, or a charlatan. His rapport with the medical department is lost, and interactions in the future will be characterized by difficulty. Other undesirable end-products are loss of self-esteem of both the patient and the health-team member, lawsuits, unemployment, creation of an unnecessary reputation as "a troublemaker," production loss, additional recruitment of workers, and costly training of the replacement. Evaluating the whole man results not only in better medicine but in prolonged employment for the worker and continuity in production for the company.

**What Is Evaluation of the Whole Man?**

The whole man is he who stands behind the chief complaint, the present illness, the physical examination, the impression, the treatment, and the disposition. The backdrop behind the patient is the collection of experiences, memories, impressions, attitudes, and values resulting from all the variables found in his life. These are his culture, his period in history, his origin, his education, his mobilizable defenses against stress, his religious beliefs, his functional capacity, his family role, his community role, his hobbies, his aspirations and drives, his job status and its place in the vocational hierarchy, his inner resources, his compassion, and his ability to give and receive affection—all these are facets of the whole man.

In view of the broad differences in the origin and nurturing of workers and their skills, is it not foolish to believe that one can understand an employee-to-be, or a worker-patient, without seeing deeply into these areas of influence? "But," one will ask, "how do I go about doing this? Isn't this meddling or prying?" No, it isn't. The only persons known to the writer who look upon or speak of this process as meddling or prying are the practitioners who are unskilled in interviewing, or the employees who have not had the opportunity of reviewing their problems with professionals interested in bringing about self-understanding.

Evaluation in this broad manner is the obtainment, recording, analysis, and use of all factors which could bear a causal relationship to a person's illness. These factors include his related history, the findings on physical examination, the emotional elements of his life or his life pattern (primarily the manner in which he deals with other people), the social factors encountered in his relationships with his parents, his spouse, and his children and in his role in the community, and certain spiritual factors which go beyond his church affiliation and activity to his own personal philosophy of life. Added to these are certain data derived from his vocational experiences—the elements of initial choice of occupation, the sense of purpose and fulfillment he finds in his work, the need for geographic mobility in his job, the desire to work alone or with a group, the necessity of creating with hands or with ideas, the relationship of work to recreation, and so on, to include all the aspects thought significant by sociologists and psychologists.

To learn of these influences, it is necessary that the physican or nurse sufficiently work through his own problems or personality defects so that he, in his interviewing, can remain interested, objective, empathic, and nonjudgmental. There must be a knowledge of the techniques of interviewing so that a patient will discuss freely the items in his social and emotional lives which relate as much to his getting well as do data regarding the appearance of the injury. Is one comfortable in seeking information from a patient regarding his acceptance of an amputation, or of atrophied legs, or regarding his sexual relationships, or his use of alcoholic beverages? Frequently, in presenting a case history, a medical student will reply to an instructor's question, "Well, I didn't pursue that." Then a discussion ensues to point out that sensitive historical data often are not "pursued" only because the interviewer is uncomfortable or inexperienced in doing so. It was not a matter of saving time or wanting to learn more of other areas—it was merely that the student knew that, were difficulties to arise in the interrogation sessions, he might not be able to extricate himself. A patient wants to unload his concerns and is eager to find someone sufficiently interested so that this can be done. He is weary of being treated by the cold, hurried, apparently disinterested person, who

manifests no concern in him as a human being. He wants desperately to encounter that particular helping person who will listen and with whom he can share his problems.

If the interviewer is warm, is outgoing, is attentive, and takes his time in approaching those areas of feeling most troublesome to the patient, there should be little difficulty at all in having the patient verbalize those feelings closest to him. This he will do if the interviewer looks at the patient and not at another point in the room; if the interviewer does not check his watch every few minutes; if the interviewer does not give evidence of restlessness by tapping his fingers on the desk, or by swinging his leg, or by rocking in his chair, or by tapping his heel, or by manipulating a pencil or a ruler. There will be free discussion if the interviewer does not laugh at those points which are embarrassing to the patient or which show up chinks in the patient's judgment or sense of discrimination. If there are sudden tears, there should be a box of tissues readily available and not embarrassed deprecations of the emotional release. A patient will share those things closest to him, if the climate is such that he can expect that everything being done is *for* him and *with* him and not *to* him. The writer has experienced many situations when, in a demonstration interview setting, a teaching conference, or with a panel before a professional society meeting, the person being asked questions about himself offered psychologic material which he thought he would never disclose. But this he did, and comfortably so, and felt better on its conclusion for having done so.

In substance, an evaluation consists of seeking as much information about the patient as possible, through examination or interview, which can shed light of the predisposition to, precipitation of, or the perpetuation of his illness.

### The Seeking of Evaluative Data

It seems indicated, before discussing the additional obtainment and use of evaluation data, that one look at some more representative situations with reference to their background explanations.

PROBLEM 1.—Why did a 32-year-old custodian have recurring acute rheumatoid arthritis every few months? Further, why, in spite of his financial and intellectual inability to support a family, did he persist in having another child every nine months or so? Reason: Repeated interviews revealed that, possibly because of old rivalry situations, he had to outdo his father, who had eight children. With the birth of the patient's ninth offspring, he had no more.

PROBLEM 2.—Why did a 28-year-old chemical engineer experience for the first time an acute epidermophytid reaction on his hands which necessitated extensive dermatologic care, daily medications, and redressings and which failed to clear in spite of the strict compliance with therapeutic recommendations? Reason: The dermatitis appeared when a co-worker was promoted to section chief, a promotion believed by the patient to be wrong and actually thought coming to him. The co-worker had been identified as a brother figure, the promotion relighting old sibling difficulties. With understanding, the skin returned to its normal state.

PROBLEM 3.—Why did a 24-year-old technician experience recurring lacerations, puncture wounds, and abrasions of his fingers over a period of two years? Reason: As developed through repeated discussions, he being a diabetic and in love with a charming young girl, was concerned over a marriage which he felt he should not enter into for fear of rearing diabetic children.

PROBLEM 4.—Why did a 28-year-old attractive and efficient stenographer, when presented with a fully developed family pattern of malignant growths among near and distant relatives, equivalent to a statistical forecast of her becoming similarly ill, refuse to have any repeated physical or laboratory examinations? Reason: Not known. Denial of illness? Wish for self-destruction?

In each of these instances, there were clinical manifestations of underlying emotional and interpersonal difficulties. If the somatic approach solely were to be followed in therapy, the cause of the persistence of these difficulties would not be disclosed. The point to be emphasized in this comprehensive approach, particularly in the diagnostic, therapeutic, and supportive activities with patients who have sustained occupational injuries, is that it is much more helpful to find out *why* a traumatic event might have taken place than *how* it occurred. Yet, in the recording of all the background information connected with an injury, we expend far too much effort on acquiring a knowledge of the events of the injury-producing accident without learning of (a) the events in the patient's life the week, day, or hour preceding the accident, (b) his mood on coming to work that day, (c) his general health that day, (d) his belief as to the cause of the injury, (e) the status of his family, (f) any anxieties which appeared recently, (g) any possible similarity between this injury and others he had sustained, (h) any resemblance to injuries incurred by his parents, siblings, or family, (i) any significance to the month, day of the week, or the hour of the trauma, or (j) any importance in the particular site of the injury.

Were one to rely more upon the answers to these kinds of questions than upon the replies to "Where were you when this happened?" or

"What were you doing when this happened?" or "Who witnessed this accident?" a much more revealing set of data would be produced. The special reward of this approach is to be found in the application of the information to the prevention of further injuries. When the influencing aspects of an employee's life are known, he can be worked with through talks or discussions—more formally and needlessly termed "interviews"—to gain insight into these accident-producing states of feeling, thought, mood, and behavior. Pointing up the need for this concept of comprehensive medicine in the better understanding of patients, Schottstaedt[1] has reminded us that, "The concept of comprehensive medicine begins with the viewpoint that all things in life interact, that disease does not occur in isolation and cannot be understood when removed from the content of the situation in which it occurs." This point is clarified further by Plummer and Hinkle[2] who, in their study of sickness absenteeism, felt that, "A great hazard in reaching a medical opinion is to consider each cold, headache, fatigue state, or gastrointestinal upset separately and independently and not to appreciate that it is part of a chronic and repetitive disease having to do with the reactions of the particular person to situations and stress of life."

No patient-employee, when seen in the industrial dispensary or in the office of the consulting surgeon, can be viewed as the possessor of a single clinical entity unrelated to the life events which he experiences every day, day after day, in a continuum.

The application of this orientation does not ease when the first visit or two has been completed but remains operational for the duration of the patient's illness or injury. The progress made by the body's reparative processes can be influenced by the effect the injury has had on the patient's family, by the promptness with which supportive disability payments are made, by the discomfiture resulting from visits to the hospital or physician's office, by the temporary loss of the traditional role of the male who leaves for work every day and now is at home, by the patient's ability or inability to follow instructions, by his depressed threshold for discomfiture, and by a host of factors both anticipated and unexpected. There must be constant feelers as to the status of the employee's situation at home, at work, or in the community subsequent to his injury. A follow-up must consist of more than the usual question of "How are you?" and the treatment indicated for that day, with the frequent disinclination to place full faith in the patient's reply.

If new anxieties arise during the course of the condition, some of the earlier exploratory questions must be placed once again to determine what environmental changes have taken place to produce these new feelings. Labile as emotional states are, no one ever can assume that a person's response to his injury will be unvarying or that the effect of his environment will remain unaltered during the convalescence.

When dealing with industrial workers who work primarily with their hands in unskilled, semiskilled, or even skilled jobs, it must be recalled that, in view of the suddenness of changes in company missions, of the quick cancellations of defense contracts because of obsolescence in methodology or product, and of the rapid substitution of automated processes for manual systems, a worker away from his job is worried and anxious. Will his job be there a few weeks from now when he is able to return? Will his wages be the same? Will he be moved to another department where he will have to establish new relationships? Will he be transferred to a new plant? Will his worth continue to be realized? These are questions which plague a person whose illness or injury has caused his being away from his accustomed work site. He has heard rumors of employees who have been kept on only during the period of the illness and then find their employment terminated on return to work. He knows of managerial personnel who believe that any person who is sick or has been involved in an accident is, by his definition, a slacker or a troublemaker. If it is possible to effect a constant communication in two parallel lines between the physician and the patient—one, regarding the clinical progress he is making, and the second, the maintenance of his job, with no change affecting him—the patient will do well and will recover in the expected length of time. Everyone needs these two elements of security; the assurance that the rate of healing matches that of past medical experience for comparable conditions, and that there has been influence on his job because of the enforced illness-absence. The practitioner who is "organ-system oriented, or technically-mechanically oriented" only will render the employee a disservice and will find an unusual prolongation of the rehabilitation time for which, in his short-dimensioned system of viewing patients, he will blame the patient, further adding worry to the already distraught worker.

The physician who lacks human understanding and the ability to recognize emotional factors in the patient's distress further will vitiate his treatment by forgetting, or not knowing,

that certain syndromes or disease entities always have emotional components which influence the course of the disease more than do treatment measures. Reference is made to the broad group of dermatoses seen among industrial workers, irrespective of the occupational or nonoccupational genesis of the skin change. Rare is the disease of the integument which is not made worse (if not caused) by stresses to which the patient has been unable to adjust. The diabetic and the person with the convulsive disorder or the rheumatoid arthritic joint or bronchial asthma is going to follow a different course when he is injured or ill than the employee unafflicted by those conditions. These well-recognized ties must be known and *accepted* by the practitioner for him to do a good piece of work in aiding the patient's recovery. This demands self-understanding by the physician, the ability to "feel for" the patient during his illness trial and not to condemn the patient for failing to handle those elements of his condition which are beyond his control. As Braceland[3] has mentioned, the "scientific and empathic understanding of illness are not mutually exclusive," and scientific medicine and a humanistic approach effectively can be combined. Without these dual methods in therapy, a part of the patient only will be treated.

### The Whole Patient Receives the Whole Treatment

In a sense, these few remarks to follow do not fit the template of what has come before. As we are discussing attitudes, though, they can be viewed as relevant. Frequently, when a worker is injured, and there are overtones of claims, suits, settlements, union intervention, court appearances, and a general aura of distasteful fractures in relationships, the physician becomes angry or apprehensive or both, and, so not to involve himself too deeply in a mire wherein he would be uncomfortable or inadequate, he carries out the minimum in care. Not all the radiographic studies are made; a complete examination is omitted; the conversation is shortened; the patient's care is rendered more by a nurse than the physician; or the patient is chastised for having been "careless" or for "not knowing better." These acts will strengthen the patient's resentment and will reinforce any thought he might have about "getting his just due" or "getting even."

This can be avoided easily, and, in addition, firm relationships can be cemented with such an employee if, from the first bit of medical care given, the absolute maximum in thoughtful attention can be provided. If all of the necessary x-ray studies are made, if the needed number of quality consultations are procured, and if as much time as needed is taken to keep the patient *au courant* in his progress through explanations neither too technical nor too patronizing, he becomes disarmed and begins to feel better. A feeling of warmth and confidence surely will reflect itself in his attitude toward the company as a whole and his own relationship and loyalty to it.


### Summary

1. Even though scientific thought has plunged farther into the cellular and molecular matter of man, it is the totality of the person in medical need which requires a full survey by the physician.

2. Illnesses and injuries are caused by a multiplicity of agents, and the interplay of physical, social, and emotional factors is seen repeatedly in the conditions presented to the occupational health team.

3. Evaluation of the whole man results not only in better medicine but in prolonged employment for the worker and continuity in production for the company.

4. The holistic evaluation of the patient is carried out through the obtainment of all pertinent social, cultural, mental, emotional, family, community, recreational, occupational, and spiritual information possible and the synthesizing of this into a full understanding of the worker's illness in the light of these factors.

5. A knowledge of interviewing techniques is required to establish an effective rapport with the employee-patient; hence, specific causally related stresses can be shared with the physician.

6. In seeking background information to an occupationally incurred injury, it is much more productive to attempt to learn from the patient *why* rather than *how* he was involved in the accident.

7. The concept must be held by all who are in medical touch with workers that no illness or injury can be viewed as an isolated event out of the context of the person's total life pattern.

8. Anxiety arising during the course of a clinical event or its rehabilitation should be explored for the possible entry of new stresses into the illness continuum so that full comprehension of the reaction pattern can be had and environmental modifications effected accordingly.

9. There must be constant communication with the employee-patient during the injury or illness period so that his sense of security can be maintained.

10. When the maximum of understanding, compassion, communication, and medical care are given the worker in need, there will be no cause for suit for nothing required has been omitted, and the potential claimant is disarmed of hostility and unfulfilled wishes regarding his professional attention. Protecting the patient automatically protects the company.

## Conclusions

1. The practice of comprehensive medical care with the industrial employee in the sense of a full understanding of all causal relationships, is mandatory for better relationships among managerial, working, and medical personnel.

2. Evaluation of the whole man is needed for the maintenance of worker health, effective preventive medical care, diminution of costly illness-absence, continued production, and a solid economy.

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AMERICAN ACADEMY OF OCCUPATIONAL MEDICINE

will hold its

FOURTEENTH ANNUAL MEETING ON FEBRUARY 7, 8, AND 9, 1962

PITTSBURGH HILTON HOTEL, PITTSBURGH, PA.

The program of scientific presentations will be as follows:

WEDNESDAY, FEBRUARY 7

Morning—George F. Wilkins, Chairman.
Topic: Executive Health Examinations

1. "Business Objectives and Procedures"—Harry B. Burr, Administrator of Medical Services, Westinghouse Electric Corp., Pittsburgh, Pa.
2. "Chronic Disease Research Opportunities"—James P. Dunn, M.D., Dr. P.H., Assistant Professor of Epidemiology, University of Pittsburgh
3. "Rectosigmoidoscopic Findings on Symptomless Examinees"—Richard L. Wechsler, M.D., Clinical Assistant Professor of Medicine, University of Pittsburgh
4. "Effect of Management Environment on Management Health"—Adolph G. Kammer, M.D., Professor of Occupational Health, University of Pittsburgh

Afternoon—Tours

1. Aliquippa Works of Jones & Laughlin Steel Corp.—Joseph L. Tauber, M.D.
2. Westinghouse Test Reactor Center (Waltz Mill, Pa.)—Albert A. Spritzer, M.D.
3. Department of Occupational Health, Graduate School of Public Health, University of Pittsburgh—Adolph G. Kammer, M.D.

THURSDAY, FEBRUARY 8

Morning—William F. Ashe, M.D., Chairman.
Topic: Hematologic Injury

1. "Heredity and Chemically Induced Anemia"—Wallace Jensen, M.D., Associate Professor of Medicine, University of Pittsburgh
2. "Metabolic Abnormalities of the Erythrocyte in Lead Intoxication"—John Harris, M.D., and Robert Griggs, M.D., Western Reserve University, Cleveland, Ohio
3. "Cytogenetics as a Tool in Hematology"—Niel Wald, M.D., Associate Professor of Radiation Health, University of Pittsburgh

Afternoon—John L. Norris, M.D., Chairman.

1. "Cardiac First Aid"—William B. Kouwenhoven, Dr. Engineering, Professor of Engineering (retired), The Johns Hopkins University, Baltimore, Md.
2. Gehrmann Lecture—Robert J. Anderson, M.D., Assistant Surgeon General, U.S. Public Health Service, Washington, D.C.

FRIDAY, FEBRUARY 9

Morning—Robert B. O'Connor, M.D., Chairman.
Topic: What Does Psychology Do For Industry?

1. "Psychological Appraisal for Selection, Placement, and Development"—Dora F. Capwell, Ph.D., Director, Psychological Service of Pittsburgh
2. "Attitudes, Morale, and Motivation"—Ray C. Hachman, Ph.D., Research Director, Psychological Service of Pittsburgh
3. "Executive and Employee Counselling"—J. Richard Porter, Ph.D., Associate Director, and John T. Bair, Ph.D., Chief, Counseling and Clinical Services, Psychological Service of Pittsburgh
4. "Research in Industrial Psychology"—Arthur W. Ayers, Ph.D., Administrator of Employment Practices, Westinghouse Electric Corp., Pittsburgh, Pa.

A varied program of events for the ladies during the meeting will include tours of the Homestead Plant, U.S. Steel Corporation; the University of Pittsburgh area, and downtown Pittsburgh, including the Old Fort and the new Civic Auditorium.

# Human Exposure to Carbon Tetrachloride Vapor

## Relationship of Expired Air Concentration to Exposure and Toxicity

**Richard D. Stewart, M.D., Ann Arbor, Mich.; Harold H. Gay, M.D.; Duncan S. Erley, M.S.; Carl L. Hake, Ph.D., and J. E. Peterson, M.S., Midland, Mich.**

CARBON tetrachloride ($CCl_4$) has continued to enjoy popularity as a solvent and chemical intermediate despite the serious consequences which may follow accidental overexposure. Wherever practical, air-monitoring devices have been used to measure the $CCl_4$ concentration in the working environment so as to reduce the risks of unrecognized overexposure. When overexposure is suspected to have occurred and the vapor concentration during the exposure is unknown, however, the immediate clinical estimation of the magnitude of the exposure is apt to be unreliable. In these circumstances, important intrinsic causes for syncope or coma may be overlooked while the patient is being treated for supposed overexposure to $CCl_4$, where actually a serious exposure may not have occurred. Thus, there exists a real need for postexposure methods for determining the magnitude of a $CCl_4$ vapor exposure.

In an effort to develop such a method, human volunteers were exposed to known concentrations of $CCl_4$ vapor and the concentration of $CCl_4$ in their blood, urine, and expired air was followed by infrared techniques described previously.[1] It was hoped that one of these analyses could be correlated with the magnitude of the exposure, with the physiological responses to the exposure, or with changes in the clinical laboratory studies induced by the exposure. The infrared analysis for $CCl_4$ in the postexposure expired air provided the sought-for method of estimating the magnitude of a vapor exposure after its occurrence and is the main subject of this paper. The concentration of $CCl_4$ in the blood and urine was too low at these exposure concentrations to be detected by the infrared method. A description of the infrared analytical methods for the determination of $CCl_4$ in expired air and a summary of the physiological and clinical laboratory data obtained from the exposures complete this presentation.

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### Experimental Procedure

*Exposure Conditions*—A tabular summary of the conditions for the three group exposures to $CCl_4$ vapor is presented in Table I. Vapor concentrations of 10 ppm were chosen for the three-hour exposures because the studies by Adams and others[2] indicated that for daily exposures of seven to eight hours' duration the average time-weighted vapor concentration should not exceed 10 ppm. The same studies indicated that the maximum instantaneous concentration should not exceed 25 ppm. The physician, however, is often confronted with cases of a brief overexposure; hence, a 70-minute exposure to 49 ppm was performed.

*Material Used*—The $CCl_4$ used in these experiments was commercial grade material with a purity of 99.0%

*Exposure Technique*—An interior room measuring 11 × 12 and 7½ foot high served as the exposure chamber. The room had one door and an air supply and exhaust system. The exhaust grill and door were closed during the experiments, but the supply grill was left open. After the subjects were seated in the closed room, $CCl_4$ was poured into a 90mm crystallization dish, which was then covered

*Table I—Human Exposure to Carbon Tetrachloride Vapor*

| Experiment | No. of Subjects | Total Exposure in Minutes | Concentration* First 5 Min. ppm | Concentration* Time remaining, ppm | Total Exposure Concentration,* ppm | Range After First 5 Min, ppm |
|---|---|---|---|---|---|---|
| 1 | 6 | 70 | 17 | 50 (Davis halide) | 49 | 31 -87 |
| | | | | 49 (Silica gel) | | |
| 2 | 6 | 180 | 6.2 | 10.9 | 10.9 (Davis halide) | 10 -14.2 |
| | | | | 11.6 | 11.6 (Infrared) | 9.5-16 |
| 3 | 6 | 180 | 11 | 10.1 | 10.1 (Davis halide) | 9 -14 |

*Time-weighted average concentration

with a folded towel. A pedestal fan blew air across the dish circulating the vapor throughout the room.

Air was sampled through 1/8 inch probes of Saran tubing suspended from the ceiling to head height at the center of the room. These probes were passed under the door to continuous monitoring devices outside the room. The $CCl_4$ vapor concentration was controlled by regulating the thickness of toweling covering the dish (Table I).

*Method of Exposure Chamber Vapor Analysis*—Three methods were utilized to measure $CCl_4$ vapor concentrations in the exposure chamber. In Experiment 1, a Dow modified Davis Halide meter[3] was coupled to a recorder which furnished a continuous record of the chamber vapor concentration. Five minutes after the exposure had started, samples were adsorbed on silica gel with subsequent combustion and analysis by Volhard titration.[4]

In Experiment 2, in addition to the continuous monitor with the Davis Halide meter, a Perkin Elmer Model 12C infrared spectrometer equipped with a 10 meter path length gas cell was coupled to a recorder and furnished a second record of the chamber vapor concentration.

In Experiment 3, the Davis Halide meter alone was used to measure the vapor concentration.

*Subjects*—Healthy male volunteers from The Dow Chemical Company ranging in age from 30 to 59 years were selected for the exposure studies. Each subject had received a complete preemployment history and physical examination, chest x-ray study, and in some cases lumbar spine x-ray film, complete blood count, urinalysis, and Orthorater eye examination. Each subject had then been followed by the medical department with periodic history and physical examinations, which included chest x-ray film, complete blood count, urinalysis, Orthorater check, and, in recent years, timed vital capacity and audiometric examination. Only one subject had an abnormal physical finding, a 59-year-old man with the liver palpable 4 cm below the right costal margin but no history of liver disease and liver function tests within the normal range.

*Frequency of Exposure*—Exposures to $CCl_4$ vapor were conducted four weeks apart. Several of the same subjects participated in the three $CCl_4$ exposures.

*Preexposure and Postexposure Data*—Immediately before each exposure the following data were obtained from each subject: blood pressure, expirogram (timed vital capacity), serum glutamic oxaloacetic transaminase (SGO-T), and urinary urobilinogen. Preexposure blood, urine, and expired-air samples were analyzed for $CCl_4$ for control purposes. Serum iron, 15-minute phenolsulfonphthalein excretion, and urinalysis were determined both before and after one of the exposures (Table II).

During the exposures, serial urine and blood samples were obtained for analysis for $CCl_4$. Subjective responses were noted. Romberg and heel-to-toe tests were performed at 15-minute intervals by those in vapor concentrations of 49 ppm.

Following the exposures, serial samples of blood, urine, and expired air were analyzed for $CCl_4$. Preexposure laboratory tests were repeated at appropriate intervals (Table II). Expirograms, urinalyses, and blood pressures were obtained within 5 to 10 minutes following the exposure. At 9 a.m. on the morning of the 49 ppm exposure, serum iron concentration was determined. This determination was repeated at 9 a.m. for the succeeding three days.

*Analysis of Blood, Urine, and Expired Air for Carbon Tetrachloride*—Venous blood was obtained through an indwelling siliconized 20-gauge needle equipped with a siliconized stylet. To a 4-ml aluminum-capped glass vial containing 2 ml of carbon bisulfide and 1 drop of 0.83% heparin solution was added 2 ml of

*Table II—Carbon Tetrachloride Preexposure and Postexposure Data*

| | | Serum Iron | | | | Serum Transaminase | | | | | Urinary Urobilinogen | | | Urinalysis: Preexposure | | Urinalysis: Immediate Postexposure | | 15 min P.S.P. | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Preexposure | 20 hr post | 44 hr post | 68 hr post | Preexposure | 20 hr post | 44 hr post | 68 hr post | 7 day post | Preexposure | 20 hr post | 7 day post | Albumin | Microscopic | Albumin | Microscopic | Pre-exposure | 20 hr post |
| Normal Value | | 9 a.m. 70-150 μgm% | | | | 6-40 units | | | | | 1:40 or less | | | | | | | 25% or greater | |
| Exp. 1, | 1 | 80 | 100 | 100 | 80 | 18 | 20 | 16 | 18 | 20 | 1:10 | 1:10 | 1:10 | neg. | 6 rbc/10 hpf | neg. | 2 rbc/10 hpf | | |
| 49 ppm | 2 | | | | | 16 | 19 | | | 20 | 1:10 | 1:10 | 1:10 | neg. | 6 rbc/10 hpf | neg. | 2 rbc/10 hpf | | |
| 70 min | 3 | 80 | 50 | 40 | 100 | 18 | 17 | 13 | 16 | 20 | 1:10 | 1:10 | 1:10 | neg. | 2 rbc/10 hpf | neg. | 0 rbc/10 hpf | | |
| | 4 | 130 | 80 | 70 | 90 | 16 | 18 | 16 | 14 | | 1:10 | 1:10 | 1:10 | neg. | 10 rbc/10 hpf | neg. | 2 rbc/10 hpf | | |
| | 5 | 110 | 60 | 120 | 100 | 20 | 22 | 23 | 16 | 20 | 1:10 | 1:10 | 1:10 | neg. | 7 rbc/10 hpf | neg. | 6 rbc/10 hpf | | |
| | 6 | | | | | 15 | 16 | | | 22 | 1:10 | 1:10 | 1:10 | neg. | 3 rbc/10 hpf | neg. | 2 rbc/10 hpf | | |
| Exp. 2, | 1 | | | | | 17 | 20 | | | | 1:10 | 1:10 | 1:10 | | | | | 36 | 39 |
| 11 ppm | 2 | | | | | 14 | 14 | | | | 1:10 | 1:10 | 1:10 | | | | | 40 | 36 |
| 180 min | 3 | | | | | 15 | 14 | | | | 1:10 | 1:10 | 1:10 | | | | | 40 | 39 |
| | 4 | | | | | 16 | 18 | | | | 1:10 | 1:10 | 1:10 | | | | | 43 | 38 |
| | 5 | | | | | 17 | 16 | | | | 1:10 | 1:10 | 1:10 | | | | | 32 | 30 |
| | 6 | | | | | 14 | 14 | | | | 1:10 | 1:10 | 1:10 | | | | | 44 | 48 |
| Exp. 3, | 1 | | | | | 18 | 15 | | | | 1:10 | 1:10 | 1:10 | | | | | | 37 |
| 10 ppm | 2 | | | | | 14 | 14 | | | | 1:10 | 1:10 | 1:10 | | | | | | 35 |
| 180 min | 3 | | | | | 14 | 14 | | | | 1:10 | 1:10 | 1:10 | | | | | | 38 |
| | 4 | | | | | 13 | 14 | | | | 1:10 | 1:10 | 1:10 | | | | | | 35 |
| | 5 | | | | | 16 | | | | | 1:10 | 1:10 | 1:10 | | | | | | 30 |
| | 6 | | | | | 17 | 13 | | | | 1:10 | 1:10 | 1:10 | | | | | | 40 |

blood. The sample was gently agitated by end-over-end inversion for five minutes. The solvent was withdrawn and analyzed for $CCl_4$ in a double-beam infrared spectrometer.[5] This method for determining organic compounds in biological fluids has been described previously in detail.[1] Voided urine samples were analyzed in a similar manner. The minimal amount of the compound detectable in blood and urine by this solvent extraction technique was 5 ppm with an accuracy of ± 1 ppm.

Expired-air samples were collected in 6-liter volume Saran bags. The bagged air was allowed to flow into the evacuated 10-meter path-length gas cell and was scanned on the Model 12C spectrometer. The absorbance at 12.6μ was measured to determine the concentration of $CCl_4$. Standard mixtures were prepared by vaporizing a weighted amount of liquid $CCl_4$ into a known volume of air. Standard mixtures were also checked by combustion and Volhard titration. The minimal amount of $CCl_4$ detectable in expired air was 0.2 ppm with an accuracy of ± 0.1 ppm.

## Results

*Experiment 1*—Six subjects were exposed to $CCl_4$ vapor, 49 ppm, for 70 minutes. During this interval, all subjects smelled a sweetish, not unpleasant odor. There was no eye or soft palate irritation. No one experienced nausea or lightheadedness. The Romberg test and the heel-to-toe testing remained normal during the exposure.

The mean and the range of the expired-air concentrations following the exposure are graphed on log-log paper in Fig. 1.

Carbon tetrachloride was not detectable in

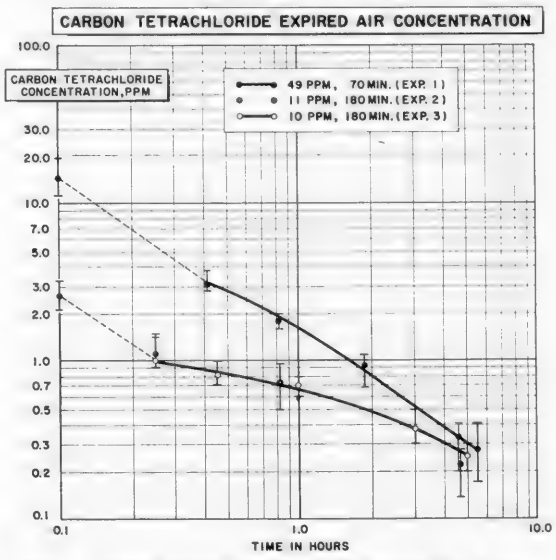


**Fig. 1—Carbon tetrachloride concentrations in expired air following exposure to vapor. Mean concentration and range are plotted.**

the blood or urine of the subjects during or following the exposure. The limit of detection was 5 ppm.

Preexposure and postexposure laboratory data are listed in Table II. The 9 a.m. serum iron concentration of two of four subjects studied showed a significant depression within the first 48 hours following exposure. One subject had an elevated urinary urobilinogen on the seventh postexposure day. No change occurred in the blood pressure or timed vital capacities of the subjects.

*Experiments 2 and 3*—Six subjects were exposed to $CCl_4$ vapor 11 ppm for 180 minutes and one month later to 10 ppm for 180 minutes. During these exposures, the subjects noted no untoward physiologic effects. The mean and the range of the expired-air concentrations following the exposures are graphed in Fig. 1. Carbon tetrachloride was not detectable in the blood or urine of the subjects during or following the exposures. The preexposure and postexposure laboratory data are listed in Table II. No change occurred in the blood pressure or timed vital capacities of these subjects.

**Discussion**

This experimental study demonstrates that the postexposure infrared analysis of expired air affords the physician a method whereby he may (1) unequivocally identify $CCl_4$ in the expired air and (2) estimate the magnitude of the exposure. The positive identification of $CCl_4$ is possible because of its characteristic infrared spectrum. This identification can be of both diagnostic and medicolegal value.

A study of Fig. 1 reveals several salient points. The $CCl_4$ concentration in the expired air decreases exponentially with time following exposure. During the first postexposure hour, this concentration appears to be directly related to the concentration of the $CCl_4$ vapor to which the subject was exposed. From these limited studies, it seems that the rate of decay of the $CCl_4$ concentration in the expired air is inversely related to the duration of the exposure. The close agreement between the 10 ppm and 11 ppm exposure data demonstrates that the infrared method has good reproducibility. $CCl_4$ determinations on expired-air samples obtained 15 minutes or more after exposure follow a smooth "decay curve" and are probably more valuable in estimating the exposure concentration than samples taken immediately after exposure, which may be contaminated with environmental $CCl_4$ vapor.

Obviously, the data presented are applicable only for estimating the magnitude of *one-* or *three-hour* exposures to $CCl_4$. It is now desirable that expired air data from experimental animals, exposed for a sufficient number of time intervals, be obtained and correlated with the existing human data. Such a body of data should provide a practical basis for estimating the magnitude of exposure to $CCl_4$ vapor.

The collection of the expired-air sample in a Saran bag prior to analysis places the use of this method in the hands of every physician. The Saran bag may be sealed and sent to a laboratory where infrared facilities are available. As only 5-10 minutes are required for analysis, the speed of reporting should be rapid and the laboratory fee reasonable. This infrared method has proven equally useful for the identification and analysis of other chlorinated hydrocarbons in the expired air[6-8] as well as for other volatile compounds possessing strong infrared absorption bands.[9]

It is not surprising that the infrared method here employed failed to detect $CCl_4$ in the blood and urine specimens. The study by McCollister and others[10] revealed that the concentration of radioactive $CCl_4$ in the blood and the urine of monkeys following vapor exposure to 46 ppm was in a range below the sensitivity of this method.

A depression of the serum iron in two of four subjects studied and an elevation of the urinary urobilinogen in one subject following the exposure to 49 ppm suggests that a brief exposure to a relatively low concentration of $CCl_4$ may be capable of altering normal metabolism in some persons. Transient depression of the serum iron 24 hours following $CCl_4$ exposure followed by elevation to twice normal levels 72 hours following exposure has been observed in beagle dogs studied in this laboratory. In this study of dogs, the serum iron fluctuations were noted to be a more sensitive index of liver insult than transaminase determinations. It is conceivable that changes in the serum iron concentration in man also may result from minimal liver insult.

Since $CCl_4$ or metabolites thereof circulate in blood for weeks following brief exposures to 40-50 ppm,[10] the initial short exposure may cause a chronic chemical stress to the liver. This could explain the elevated urinary urobilinogen noted seven days after exposure in one subject. Elevated urinary urobilinogen levels may be observed in the 7- to 14-day interval following exposure to other chlorinated hydrocarbons.[6, 11]

In the past four years, this medical department has found the urinary urobilinogen determination to be a more sensitive index of liver insult from chlorinated hydrocarbons than either of the two transaminase determinations.

## Summary

A series of three controlled human exposures to carbon tetrachloride vapor was conducted. The infrared analysis of the postexposure expired air was demonstrated to be a practical method with which to estimate the magnitude of the exposure. Physiological and clinical laboratory studies were carried out in conjunction with the three vapor exposures. Postexposure depression of the serum iron concentration and elevation of the urinary urobilinogen were noted in some of the subjects following exposure to $CCl_4$, 49 ppm, for 70 minutes.



## Acknowledgment

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(Requests for reprints should be addressed to the Biochemical Research Laboratory, The Dow Chemical Company, Midland, Mich.)


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# Studies of the Concentrations of Chromium Salts in the Skin of Guinea Pigs

M. H. Samitz, M.D.; Sidney Gross, M.D.; and Peter Flesch, M.D., Philadelphia, Pa.

IN A PREVIOUS paper[1] we reported on the influence of sodium lauryl sulfate on sensitization of guinea pigs to chromium salts. We were unable to confirm previous findings of induced sensitization on challenge testing.[2] The present paper deals with further experiments supporting these preliminary results.

### Attempts to Induce Sensitization with Various Concentrations of Potassium Dichromate

*Experimental Procedure*—(a) On the shaved left flank of six guinea pigs, 0.5 ml. of 1% sodium lauryl sulfate was rubbed with a glass rod for 30 seconds. Immediately thereafter 0.5 ml of 0.5% potassium dichromate was rubbed into the same area for 30 seconds. This procedure was repeated daily for eight days.

After eight days there was no reaction at the sites of application.

Challenge doses of 0.5 ml of 0.5% potassium dichromate were applied to the shaved right flanks of these guinea pigs two and four weeks later. In no case was there a reaction to indicate sensitization.

(b) Since we had observed irritating reactions produced by 5% and 10% potassium dichromate, we considered the possibility of inducing sensitization with higher concentrations. For this purpose 5% potassium dichromate was applied to six guinea pigs in the same way as described above under (a).

By the third day, all animals showed erythema and scaling. In two animals, there was ulceration by the eighth day.

Challenge doses of 0.5 ml of 0.5% potassium dichromate, applied at two and four week intervals to the shaved flanks of the guinea pigs, produced no reaction in any of the animals.

### Absorption of Hexavalent and Trivalent Chromium in the Skin of Guinea Pigs

In our previous study[1] we demonstrated the percutaneous absorption of chromates by spectrographic analyses of various animal tissues. These experiments were extended by introducing a new technique of tattooing, to insure deposition of the chromium solution in the skin. In order to compare the influence of the valence of chromium upon its diffusibility, both potassium dichromate and chromium nitrate were used. Hueper and Payne[3] have shown that trivalent chromium is strongly bound to proteins, while hexavalent chromium is attached to a slight extent and in an unstable way. Samitz and Gross[4] expressed the belief this finding could explain the release of solubilized hexavalent chromium by sweat from chrome tanned leather.

*Experimental Procedure*—(a) Ten guinea pigs were tattooed on the shaved flanks with

*Table I—Chromium Content* in Wet Tissue Specimens of Guinea Pigs Tattooed with 0.5% Potassium Dichromate*

| Guinea pig | Time of Observation 24 hr | 48 hr | 96 hr | 1 week |
|---|---|---|---|---|
| 1 | 5.9 | 2.4 | No specimen | 0.0 |
| 2 | 0.7 | 0.4 | 1.2 | 0.1 |
| 3 | 0.1 | 0.4 | 0.1 | 0.1 |
| 4 | 1.8 | 0.6 | 0.4 | 0.1 |
| 5 | 4.1 | 0.9 | 0.9 | 0.6 |
| 6 | 4.7 | 1.5 | 0.3 | 0.1 |
| 7 | 1.5 | 0.9 | 0.1 | 0.5 |
| 8 | 1.2 | 0.5 | 1.2 | 0.6 |
| 9 | 1.5 | 0.9 | 1.5 | 1.8 |
| 10 | 1.5 | 0.6 | 0.0 | 0.9 |
| Average | 2.3 | 0.9 | 0.6 | 0.5 |

*Values are expressed as ratios of chromijm found to chromium used in tattooing $\times 10^2$.

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*Table II—Chromium Content* in Wet Tissue Specimens of Guinea Pigs Tattooed with 0.5% Chromic Nitrate*

| Guinea pig | Time of Observation 24 hr | 48 hr | 96 hr | 1 week |
|---|---|---|---|---|
| 1 | 0.3 | 4.6 | No specimen | 0.0 |
| 2 | 3.8 | 5.4 | No specimen | 0.3 |
| 3 | 1.5 | 4.6 | 0.6 | 0.0 |
| 4 | 0.3 | 0.1 | 4.6 | 0.1 |
| 5 | 1.5 | 3.0 | 0.0 | 0.0 |
| 6 | 1.8 | 1.5 | 3.0 | 1.8 |
| 7 | 7.7 | 3.0 | 0.3 | 0.0 |
| 8 | 0.4 | 0.8 | 0.0 | 0.0 |
| Average | 2.2 | 2.9 | 1.4 | 0.3 |

*Values are expressed as ratios of chromium found to chromium used in tattooing $\times 10^2$.

1 ml of 0.5% potassium dichromate (corresponding to 1,700 $\mu$g $Cr_{VI}$) over an area 3 cm in diameter. Punch biopsy specimens (7 mm) were taken from the central portion of the 3 cm tattooed areas at 24, 48, 96 hours and one week. The tissue specimens were analyzed spectrographically for chromium (Table I).

(b) Eight animals were tattooed with 1 ml of 0.5% chromic nitrate (corresponding to 650 $\mu$g $Cr_{III}$) as in (a). The results of spectrographic analyses are shown in Table II. Spectrographic analyses of tissue tattooed with water in two control animals showed no chromium. The amount of chromium in the skin showed no consistent relation to the valence of chromium compound.

## Summary and Conclusions

There was no evidence of epidermal sensitivity on challenge testing in animals treated with 0.5% potassium dichromate in combination with sodium lauryl sulfate. Although a 5% potassium dichromate solution in combination with sodium lauryl sulfate produced marked irritation, sensitivity was not demonstrated. These results confirm our previous findings.

Deposition of hexavalent and trivalent chromium solutions in the skin was obtained by tattooing. Spectrographic analyses of biopsy sections did not show any significant difference in the absorption of two types of chromium compounds. After one week, little of either compound remained in the skin.

(Dr. Samitz' address is 1715 Pine St., Philadelphia 3, Pa.)

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# Fluorides: A Critical Review

## V. Fluoride Intoxication in Laboratory Animals

Robert K. Davis, Ph.D., Cincinnati, Ohio

THE PHYSIOLOGICAL characteristics of fluoride intoxication have been under investigation by toxicologists since before the turn of the century. Very early in these investigations, experimental animals were used in attempts to define the effects of the absorption of fluoride and the mechanisms of its action. In the course of these investigations two descriptive patterns of the behavior of fluoride in the animal organism have evolved, one relating to acute intoxication and the other to a chronic form of the disease.

### Acute Intoxication

The comparative toxicity of certain common fluoride compounds administered orally in one dose to various species of laboratory animals is represented by data from various sources. The solubility of the compound administered (or ingested otherwise) is, of course, an important factor in its immediate toxicity. When ingested in sufficient quantity or in sufficiently concentrated form, the fluoride ion has a direct, severely irritating effect on the gastroenteric mucosa. The degree of local irritation varies with the concentration of the compound in the solution administered or in the gastroenteric contents; high concentrations tend to induce an acute hemorrhagic gastroenteritis, while sufficiently low concentrations may exert no demonstrable immediate effect. Fatal intoxication may result from the repetitive ingestion of sublethal or even mildly toxic doses, provided the interval of time between doses of sufficient size is such that an acutely toxic concentration builds up in appropriate tissues of the body. Representative data are presented in Table II.

Signs of acute systemic intoxication are salivation, lacrimation, vomiting, diarrhea, muscular fibrillation, and respiratory, cardiac, and general depression. In fatal intoxication some or all of these signs appear in heightened intensity and terminate in clonic convulsions, collapse, coma, and death. The singular rapidity of onset and progression of the intoxication

*Table I—Immediate Toxicity of Fluoride Compounds Administered Orally to Laboratory Animals*

| Fluoride Compound | Species of Animal | Lethal Dose (mg/kg) | Reference |
|---|---|---|---|
| HF (aqueous solution) | Rat | 50 | 1 |
| NaF | Guinea pig | 50-223 | 2 |
| | | | 3 |
| | | | 4 |
| | | | 5 |
| | | | 6 |
| | | | 7 |
| | Hamster | 80* | 8 |
| | Mouse | 46* | 9 |
| | Rabbit | 200* | 10 |
| | | | 11 |
| | | | 12 |
| | | | 3 |
| | | | 13 |
| | | | 14 |
| | | | 15 |
| | | | 16 |
| | Rat (male) | 250* | 17 |
| | | | 18 |
| | Rat (female) | 180* | 19 |
| | | | 20 |
| $Na_2SiF_6$ | Guinea pig | 60-250 | 4 |
| | | | 6 |
| | | | 3 |
| | | | 21 |
| | Rabbit | 125* | 14 |
| | | | 22 |
| | | | 23 |
| | | | 24 |
| | Rat | 125* | 20 |
| | | | 25 |
| $Na_3AlF_6$ (natural cryolite) | Rabbit | (gm/kg) 9-12 | 26 |
| | Rat | 16 | 26 |
| $Na_3AlF_6$ (synthetic cryolite) | Guinea pig | 3* | 27 |
| | Rabbit | 9 | 27 |
| | Rat | 1.5 | 27 |

*$LD_{50}$

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Dr. Davis is Assistant Professor of Industrial Health, University of Cincinnati College of Medicine.

*Table II—Fatal Acute Intoxication in Laboratory Animals Following the Repetitive Administration of Sublethal Dosages of Inorganic Fluorides*

| Compound | Species of Animal | Dosage (mg/kg) | Number of Doses Administered on Successive Days | Average Length of Survival (days) | Reference |
|---|---|---|---|---|---|
| NaF | Guinea pig | 20 | 16-22 | 21 | 2 |
| | Guinea pig | 53 | 6 | 7 | 28 |
| | Guinea pig | 50/animal | 22 alternate days | 45 | 3 |
| | Rabbit | 100 | 3 alternate days | 6.3 | 29 |
| | Rat | 50 | 3 | 3 | 17 |
| $Na_2SiF_6$ | Guinea pig | 100/animal | 4 | 4 | 21 |
| $Na_3AlF_6$ (natural cryolite) | Rat | 1,500 | 20-52 | 20-52 | 30 |
| $Na_3AlF_6$ (synthetic cryolite) | Rat | 8,000 | 10 | 6 | 26 |

varies directly with the magnitude of the initial dose. Not all of the signs listed are observed in all cases, nor do they occur necessarily in the order designated. The anatomic lesions of fatal acute intoxication are nonspecific, but the evidence of gastroenteric irritation are more general and more intense than those found, as a rule, in most of the other forms of gastroenteritis. Other characteristic findings are acute hyperemia of all of the viscera and varying degrees of cloudy swelling, softening, and degeneration with varying degrees of necrosis of the liver and kidneys, with degenerative changes of lesser degree in the heart and brain.

Acute intoxication may also result from single or multiple episodes of respiratory exposure to elemental fluorine and to certain inorganic compounds of fluorine, as hydrogen fluoride and silicon tetrafluoride. Illustrative data are given in Tables III and IV. These data and the comments which follow are included to call attention to certain properties of elemental fluorine, which occurs not in nature but only in connection with certain specialized technological procedures, and to those of certain other inorganic compounds of fluorine, the primary effects of which, in their usual impact on the animal organism, bear little resemblance, generally, to fluoride intoxication. These gases act primarily as severe respiratory irritants. Fluorine reacts chemically with vigor and sometimes with violence with almost every element or material, thereby severely injuring the respiratory tract. Hydrogen fluoride and silicon tetrafluoride also induce upper and lower respiratory damage, in much the same manner as acid gases. The results of respiratory exposure to these materials, as a rule, depend primarily upon the severity and extent of the direct injury to the respiratory exposure. If the pulmonary damage is not sufficient in itself to be lethal, systemic intoxication may follow. Dependent upon the concentration of the material in the respired air and the duration of the exposure, inhalation of these gases induces acute hyperemia and edema of the conjunctival and nasal mucosae, acute inflammation of the bronchial mucosa, progressing to necrosis of the epithelium and acute pulmonary edema. If the animal survives long enough, the other organs exhibit nonspecific acute hyperemia, edema, and degenerative changes.

The pathology and the pathological physiology of acute fluoride intoxication, historically, include a wide variety of abnormalities. In 1897, the injection of sodium fluoride in the dosage of 54 mg per kilogram into a loop of the small intestine of a dog was observed to have resulted in necrosis of the epithelium and the subsequent death of the animal.[34] Another investigator[35] reported that a 0.6% solution of sodium fluoride, ingested in milk by dogs, caused retarded production of gastric and biliary secretions, spastic contraction of the pylorus, and diminished gastric motility. Similar effects were observed in rats, guinea pigs, cats, and rabbits.[36-39] A comparison of the effects of oral and intraperitoneal administration of sodium fluoride revealed that certain digestive enzymes were inhibited by the oral route.[40] Radiotracer techniques have shown that fluoride is readily absorbed from the gastroenteric tract and permeates the tissues.[41] Inhibition of liver phosphatase[42] and hyperglycemia[43-45] have been demonstrated following the administration of fluorides. Estrus, reproduction, and lactation are disturbed by the oral administration of fluoride in dosages which exceed certain levels[46, 47]; the threshold for rats is 25 mg per kilogram per day, and that for guinea pigs is 10 mg per kilogram on alternate days.

The essential mechanism (or mechanisms) of acute intoxication by fluoride remains ob-

*Table III—Fatal Intoxication in Laboratory Animals from Respiratory Exposure on One Occasion to Elemental Fluorine or to Certain Volatile Inorganic Compounds of Fluorine*

| Compound | Species of Animal | Concentration (ppm by volume) | Length of Exposure (hours) | References |
|---|---|---|---|---|
| $F_2$ | Guinea pig | 200 | 3 | 31 |
| | Mouse | 200 | 3 | 31 |
| | Rabbit | 200 | 3 | 31 |
| | Rat | 200 | 3 | 31 |
| HF | Guinea pig | 250 | 3 | 32 |
| | Guinea pig | 660 | 1 | 32 |
| | Rabbit | 250 | 3 | 32 |
| | Rabbit | 660 | 1 | 32 |
| $SiF_4$ | Guinea pig | 5,000 | 0.5 | 33 |
| | Rat | 5,000 | 0.3 | 33 |

*Table IV—Fatal Intoxication in Laboratory Animals from Repetitive Respiratory Exposure to Elemental Fluorine and Hydrogen Fluoride*

| Compound | Species of Animal | Concentration (ppm by volume) | Duration of Exposure | | References |
|---|---|---|---|---|---|
| | | | Hours per Day | Consecutive Days* | |
| $F_2$ | Guinea pig | 16 | 5 | 18 | 31 |
| | Mouse | 16 | 5 | 18 | 31 |
| | Rabbit | 16 | 5 | 18 | 31 |
| | Rat | 16 | 5 | 18 | 31 |
| HF | Guinea pig | 10 | 6 | 31 | 31 |
| | Rabbit | 10 | 6 | 31 | 31 |
| | Mouse | 33 | 6 | 30 | 31 |
| | Rat | 33 | 6 | 30 | 31 |

*Excluding Saturdays and Sundays

scure. For obvious chemical reasons, reinforced by neuromuscular phenomena that bear a degree of resemblance to tetany, a gross disturbance of the calcium metabolism has been postulated as an important, perhaps *the* important, component of the action of fluoride. Evidence in substantiation of this concept is lacking, and the descriptive approach to this disease continues to be its best expression in toxicology.

### Chronic Intoxication

The chronic manifestations of the absorption of relatively small (as compared to those capable of inducing acute intoxication), but excessive, quantities of fluoride, irrespective of the mode of administration, differ descriptively from those of acute intoxication. In the young animal, there is retardation of growth and development. Sodium fluoride in the diet to the extent of 0.10% by weight, retarded the growth of adult rats and depressed the rate of growth and the length of survival of the offspring.[18, 51]

Early investigators[52-54] noted that prolonged absorption of relatively low concentrations of fluoride altered the structure of the bones.[55] Generally, there was enlargement of the long and flat bones and a subsequent interference with the functioning of joints. In the adult animal, there was thickening and increased density of the cortices of the bones.

Microscopically such bones show irregular, coarse, dense calcification along the endosteum and periosteum. The bony trabeculae are irregular and exhibit increased lines of resorption. Young animals may develop osteoporosis and exhibit wide, poorly calcified zones of periosteal bone formation. Microscopically, there is osteoblastic activity and formation of osteoid tissue which is poorly calcified.

The second major effect of the prolonged absorption of excessive quantities of fluoride by the commonly used experimental animal is that of injury to the teeth.

The literature on this facet of the subject is almost as voluminous as the combined published material on all of its other aspects. The absorption of fluoride from the diet at levels of 100 ppm or more therein by weight produces pigmentation, chalking, pitting, corrosion, and friability of the teeth. In the teeth, as in bone, an excess of fluoride causes decalcification of the dentin and enamel. Concentrations of 10 ppm (or more) in the diet result in incipient striation of the dental enamel. Levels above 300 ppm produce macroscopic hypoplasia of the enamel with pitting and corrosion.

It has generally been observed that fluoride administered in the water supply is more effective in producing the dental and skeletal alterations than similar concentrations contained in the food.[56-58]

On the other hand, the regular absorption of fluoride, within certain limits of concentration or dosage, has been found to exert a beneficial effect upon the teeth. Fluoride in the order of 1 ppm in the diet, or preferably in the water supply, effectively prevents the occurrence of dental caries in the animal. In the rat, levels up to 10 ppm in the diet were most efficacious as a caries-inhibitor. Small quantities of fluoride, as a dietary supplement, have shown beneficial effects on growth and development.[59, 60]

## Pathologic Physiology

### Heart

A group of investigators[61] fed sodium fluoride to young rats within certain limits of dosage, and recorded the effects on the heart. There was a marked regressive degeneration of the myocardial fibers (cloudy swelling and vacuolar degeneration), interstitial cellular exudate, and focal small hemorrhages. These changes occurred in one month when the diet contained fluoride in the concentration of 50 ppm, and after two or three months when the dietary level of 5 to 10 ppm was maintained. Electrocardiograms of rabbits given daily doses of 10, 30, 50 and 100 mg. per kilogram of sodium fluoride in their basal ration[62] were made. These demonstrated the occurrence of myocardial changes characterized by depressed ST and inverted waves, prolonged Q-T intervals, multifocal ventricular premature contractions, and bundle branch blocks. These phenomena correlated with anatomic lesions demonstrated by subsequent microscopic examinations of cardiac sections. The severity of these lesions varied directly with the magnitude of the dosage of sodium fluoride and the duration of its administration.

### Kidney

The kidney appears to be the most vulnerable of the organs in fluoride poisoning in the mammal. In investigations of delayed acute intoxication, this organ has been shown to be affected first and most severely.

Microscopically, the kidneys of rabbits, given 30 or 50 mg of sodium fluoride per kilogram sustained glomerular changes such as increased cellularity, capillary hyperemia, and cellular exudation. The tubules underwent epithelial degeneration, and had protein casts and erythrocytes in their lumina.[63-65]

### Endocrine glands

Quite early in the pharmacological and toxicological investigations of fluorides, interest was expressed in the effects on various mammalian endocrine glands. One investigator[66] observed, in guinea pigs and goats fatally poisoned with fluoride, that the pituitary and thyroid glands were atrophic and nonsecretory.

There appears to be sufficient evidence that high concentrations of fluoride inhibit the action of the trophic hormones of the pituitary,[67, 68] thereby retarding growth and reproduction.

Other endocrine organs investigated and found to be affected by fluoride, both histologically and functionally, were the parathyroids, adrenals, pancreas, and gonads. Investigators,[69] in attempting to elucidate the mechanism of action of hypophyseal hormones, first injected sodium fluoride intravenously into rabbits at the rate of 150-300 mg/kg over a period of 72 hours following the intravenous injection of a gonadotropic hormone. This procedure completely inhibited stimulation of the ovary by the gonadotropins without inducing histologic changes in the ovaries. However, in feeding experiments with guinea pigs, one observer[70] found ovarian congestion, arrest of follicular development, and fatty degeneration. The fluoride had been fed at the level of 10 mg/kg on alternate days for 21 days.

Another investigator[71] induced fluoride intoxication in the rat by the daily administration of intraperitoneal doses over a period of 15 days. Each rat received a total of 400 mg of sodium fluoride. On gross examination of the thyroid, the pancreas, and the adrenal glands, no abnormalities were observed, with the exception of some apparent increase in weight, in association with cortical hypertrophy of the adrenals. The follicular epithelium of the thyroid showed a reduction in the size of the nuclei and in the amount of cytoplasm, but the follicular cells remained columnar. In the pancreas, the acinar cells exhibited an increase in mitotic activity.

Early investigation of the relationships of sodium fluoride and calcium fluoride to the thyroid-parathyroid functions indicated that low doses of sodium fluoride caused hypertrophy of both glands, and that calcium fluoride produced no macroscopic or histologic changes.[72] However, calcium fluoride administered in small doses caused a generalized hypercalcification of bony structures, and sodium fluoride produced osteoporosis.

The rate of elimination of fluoride that has been deposited in bone may be influenced by the parathyroid, but the osteopathy cannot be explained solely by the action of fluoride upon these glands.

### Blood

In respect to the coagulation of the blood, varying observations and reports have been made. When small quantities of fluoride were fed to goats for 20 months, the time required for the coagulation of their blood was found to be prolonged.[73] Another observer[74] noted that in vitro additions of isotonic sodium fluoride to blood delayed coagulation, and explained this phenomenon on the basis of the precipitation of calcium fluoride. He found that the addition of sufficient calcium to this plasma, together with noncellular tissue extracts, resulted

in coagulation. Intravenous injections of 3% solution of sodium fluoride had little effect on the rate of coagulation of the blood of animals until the concentration of fluoride in the blood exceeded 45 mg %, at which level coagulation is inhibited. Young puppies given multiple oral doses of sodium fluoride sustained reduction in the rate of coagulation.[75] The same investigator reported experiments in which adult dogs were fed on diets containing fluoride throughout a year; no change in the coagulation time occurred when the dosages ranged from 0.45 to 4.52 mg per kilogram. Another investigator[76] reported that when rabbits were given quantities of sodium fluoride orally for five months, their serum calcium fell and the inorganic phosphorus rose; when calcium fluoride was given, the calcium rose and the phosphorus fell. Intravenous doses of 0.1 to 5 mg of sodium fluoride administered to 5-lb. rabbits had no effect on the coagulation of their blood, but doses of 10 to 60 mg greatly shortened the coagulation time, while doses of 200 mg, or more, prolonged the coagulation time.[77]

There are abundant experimental data[76, 78, 79] to demonstrate the calcium-precipitating action of the fluoride ion. Thus, if the serum calcium level is reduced sufficiently as a consequence of fluoride intoxication, the coagulation process will be prolonged. Conversely, if the level is increased, coagulation may be accelerated.

### Liver

The effects of fluoride on the liver have been described by many investigators.[71, 80, 81] The severity of these effects has been found to vary directly with the dosage administered to any one species of animal. The route of administration is of little importance except as it may affect the time-concentration factor of the fluoride ions reaching the liver. Lesions have been described[82, 83] ranging from mild cloudy swelling to fatty degeneration and necrosis of the parenchyma, particularly in the region of the central veins. Prolonged administration of fluoride in low dosage did not interfere with vitamin C storage in the liver in guinea pigs, rats, and cows, but did increase the vitamin C content of the adrenal and pituitary glands.

## Absorption and Distribution

An investigation of the metabolism of fluoride in rats, using radioactive fluoride, demonstrated the presence of significant quantities of fluoride in the contents of the stomach and intestine one hour after the administration of fluoride intravenously.[43] The saliva and bile contained radioactive fluoride but did not contribute significantly to its presence in the gastroenteric contents. The radiotracer was believed to have been transported from the blood into the digestive secretions by some process resembling diffusion, thus gaining entry to the gastroenteric tract. The extent to which this occurred varied directly with the concentration of radioactive fluoride in the blood, and the rate of flow of the blood through the secreting organs.

Following the intravenous injection of radioactive fluoride into rats, the kidney was found to have the greatest concentration of $F^{18}$ (10 minutes and 90 minutes after administration), and was the principal organ of excretion.[84] Shortly after oral administration, the kidney had the second highest concentration of $F^{18}$, the stomach and small intestines having first place in this regard. A sharp decrease in the quantities of fluoride in various organs of the rat occurred in the interval between the initial and later observations, at 10 and 90 minutes, respectively, and is evidence of the rapid removal of fluoride from the soft tissues and circulating blood. When renal tubular damage was induced by the administration of uranyl nitrate, little change was observed in the distribution of fluoride excreted in the urine and feces.[85] Renal clearance tests indicated that fluoride is resorbed to a much lower degree than chloride and that there was no indication of a renal tubular secretion of fluoride.

An investigation[63] of the effects upon the kidney of the administration of sodium fluoride, in doses ranging upward to 50 mg. per kilogram over periods varying from two to eight weeks, demonstrated that the plasma urea clearance was decreased. The degree of this decrease varied directly with the magnitude of the total quantity of fluoride administered (individual dose times frequency). The renal plasma flow (RPF) indicated by the clearance of *p*-aminohippuric acid and the rate of glomerular filtration (GFR) involved in the clearance of exogenous creatinine were employed to determine the effect of a slow intravenous infusion of varying quantities of 1% isotonic saline solution of sodium fluoride. Comparatively large quantities of fluoride induced a marked decrease in renal function in both instances, while those below a certain level exerted little or no effect.

It has been established[86] that fluoride ion is distributed into the fetus via the placenta, and into the bodies of nurslings via the milk of females bearing $F^{18}$.

An investigation[87] to determine the rates of absorption and distribution of sodium fluoride into the tissues of six-month-old lambs given radioactive sodium fluoride ($Na^{22}F^{18}$) by cap-

sule, demonstrated radioactivity attributable to both sodium and fluoride in the blood as early as five minutes after the administration of the compound. The initial absorption from the rumen was rapid, but most of the quantity administered remained in the rumen at the end of two hours. The fluoride activity in the blood reached its maximum in three hours following the administration of the capsule, after which it decreased progressively. Autoradiograms prepared from selected bones after the administration of a capsule demonstrated a heavy deposition of $F^{18}$ in the primary spongiosa below the epiphyseal plate, and a generalized spotty distribution in the trabecular region. There was a scattered but linear deposition of activity in the region of bone of endochondral origin, and a relatively light deposition along the endosteal and periosteal surfaces.

## Discussion

This review was initiated for the expressed purpose of correlating and bringing together experimental data on the toxicity of the fluoride ion for mammalian species. Some of the data published during the last 65 to 70 years have been of fragmentary or contradictory types, and a clear concept of the toxic effects of fluoride in the animal organism has been difficult to obtain.

The more important facts that emerge from a critical review of the literature have to do with the relationships of time, dose, and concentration to the general systemic effects of fluoride intoxication. As a corollary observation, these general systemic effects have a remarkable similarity from species to species, despite physiological differences between species. Thus the fluoride ion must exert its influence upon some basic physiologic process. Its inhibitory action on certain enzyme systems in vitro[88] suggests that it may have similar significant effects in vivo. Supporting evidence for this general hypothesis is lacking, however.

The variations in the effects of various compounds of fluorine, with specific reference to their degree of severity, have been due largely to their molecular form and to their associated solubility and rate of absorption, or in some instances (e.g., fluoborate) to a complex nondissociating radical containing fluoride. In addition, it is clearly evident that the effective level of dosage of specific compounds, in relation to specific effects, varies from one species of animal to another. No clear answer has been obtained experimentally to the question of the quantity or concentration of fluoride ion that is capable of killing or suspending the functioning of specific tissue cells. The results of tracer investigations have demonstrated that the fluoride ion passes readily into erythrocytes and other tissue cells of all species of animals.[11, 87]

The pattern of acute intoxication indicates that the central nervous system is affected very early. Specifically, following the administration and the progression of absorption of a large single dose of fluoride, the appearance of generalized nervous irritability, lacrimation, salivation, emesis, and rapid respiration indicates a central nervous stimulation. As absorption continues, these signs become severer, accompanied by a rapid fall in blood pressure and a terminal slowing and irregularity of respiration, with respiratory failure and death. If absorption is less rapid, or if the lethal dose is administered in broken doses over a considerable period of time, the animal develops a striking inanition, feeds poorly, sustains progressive loss of weight, and relinquishes life in an undramatic manner. Usually the gross and microscopic examination of such animals, post mortem, reveals little or nothing which points definitely to a specific cause of death.

The experimental investigations of the absorption, distribution, and excretion of fluoride have shown that fluoride leaves the blood and other soft tissues of the body rapidly; this results in varying degree under variable conditions, from the excretion of fluoride, mainly in the urine, and from its deposition. The soft tissues of the animal are spared thereby from prolonged exposure to high concentrations of fluoride. Such exposure is brief, indeed, considering the relatively high rate of excretion of fluoride, as compared with many other anions, as well as the speed with which deposition in the skeleton occurs. When the animal is subjected to conditions under which the absorption of fluoride is prolonged or intermittent, the level of dosage must be high, and the interval between doses must be brief if injurious effects are to be sustained by the soft tissues (i.e., the principal viscera). On the other hand, it is evident that most of the organ systems are vulnerable to the toxication of fluoride if the concentrations within them reach sufficiently high levels. The skeleton, obviously, is highly resistant to such injury; the kidney appears to occupy the opposite position of greatest susceptibility to relatively low or intermediate concentrations of fluoride.

Another matter that has been established by investigations of the toxicological effects of the absorption of fluoride by animals at various rates and over widely variable periods of time is the existence of conditions, with respect to

dosage and time, which are unattended by any harmful effects. Slightly higher rates of absorption over sufficient periods of time (at levels approaching the threshold of harmful effects) may induce a barely detectable degree of mottling of tooth enamel (unless the dosage is initiated at the proper time and in sufficient quantity, this will not occur) as the only demonstrable response of animals.

An area of research that has yet to be explored successfully is that of the mechanism of the toxic action of fluoride. That this same void exists in our understanding of many other types of intoxication is no reason for ignoring this issue.

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U. S. Atomic Energy Commission

offers

Special Fellowships in Industrial Hygiene

Fellowships in industrial hygiene provided by the Atomic Energy Commission will be given at the University of Cincinnati, Harvard University, University of Michigan, University of Pittsburgh and the Wayne State University. The tenure of the fellowships is the academic year beginning with the fall term. Applications are due March 1.

The program of study, which leads to a master's degree, is open to graduates in the physical sciences, the biological sciences (with adequate preparation in physics, chemistry, and mathematics), and engineering. The several university programs differ somewhat in detail, but all follow a common pattern. The courses of study fall into three broad groups:

1. Underlying courses in health science: biostatistics, epidemiology, general environmental health, and public health practice.
2. Health sciences pertaining particularly to occupational health problems, environmental physiology, toxicology, radiobiology, principles of industrial hygiene and industrial medicine.
3. Specific practices in industrial hygiene: physical and chemical methods of analysis and evaluation of environmental hazards, industrial ventilation, measurement and control of noise, radiation hazards, aerosol physics, air-cleaning methods.

Fellows must be United States citizens, under 35 years of age, and acceptable for graduate work at the university to which they are assigned. The basic stipend for the academic year is $2,500. An additional $350 is allowed for a spouse and $350 for each dependent child. Normal tuition and fees required by the university as well as a limited travel allowance are paid by the fellowship. Fellows having one or more years' graduate work or industrial experience in a related field may be eligible for an additional $200 in the basic stipend.

Applications must be supported by letters of reference, and transcripts. Forms may be obtained from the Industrial Hygiene Fellowship Office, Oak Ridge Institute of Nuclear Studies, Oak Ridge, Tenn. Appointments will be made on or about April 1.

# Editorial

## Some Handshakes

THE YEAR'S end finds us in a complimentary mood. As a result of hard work by a large group of capable colleagues our journal is increasingly accepted as the authoritative voice of industrial medicine. Some handshakes are in order.

First of all, we say goodbye to three members of the Editorial Board whose terms of service end with this issue. These are Drs. Russell G. Birrell, Ronald F. Buchan, and Frank Princi. They helped work over the early JOM manuscripts in the summer and fall of 1958, months before our first issue went to press, and have since that time contributed steadily, substantially, and silently to each month's product. To the well-known achievements of each in the field of industrial medicine is now added recognition of another solid contribution.

Nine new names appeared on our list of literature abstracters during the year. Robert Auerbach, M.D., Bethesda, Md., and William L. Epstein, M.D., San Francisco, Calif., joined the group covering dermatology. Robert T. Clark, Ph.D., Oklahoma City, Okla., became a contributor in environmental physiology, giving emphasis to problems in aviation. Norbert J. Roberts, M.D., New York, N. Y., accepted responsibility for heading the internal medicine literature section, which was joined by Richard W. Call, M.D., of Los Angeles, Calif. Otto P. G. Preuss, M.D., Columbus, Ohio, entered the group covering clinical toxicology, giving particular attention to articles in the German language literature. Three replacements were made in our surgery abstracts section, which brought in A. A. Bullock, M.D., Shreveport, La.; Elton Mendeloff, M.D., Milwaukee, Wis.; and H. L. Skinner, M.D., Carroll, Iowa. A welcoming handshake is extended to these new members of the groups whose monthly production of literature abstracts takes our journal a long way toward its goal of presenting the industrial practitioner with all of the basic reading material needed to stay abreast of this field of practice.

The highlight of our publication year undoubtedly was the March issue, which carried a special supplement that presented the details of a radiation fatality. The arrangements for this production were accomplished through Thomas L. Shipman, M.D., of the Los Alamos Scientific Laboratory, whose assistance throughout the total enterprise was the foundation of its success.

A major change of format in this year's American Industrial Health Conference held implications of major significance for our journal. For the first time the American Industrial Hygiene Association and the American Conference of Governmental Industrial Hygienists held their annual meetings elsewhere. Many areas covered in the scientific programs of these two organizations are of great interest to industrial physicians. In recognition of this, Jean S. Felton, M.D., who had responsibility for IMA's scientific programs at the 1961 meeting in Los Angeles, arranged sessions that dealt with industrial hygiene subject matter. Most of these papers are now scheduled for appearance in future issues of our journal. These, and the unusually high proportion of other publishable papers presented at the Los Angeles meeting, have created a greater backlog of articles than can be easily handled. At our present rate of publication, authors on the average could expect a year's wait between acceptance of an article for publication and its appearance in print. Comforting as this may have been to editorial and production personnel, the situation did call for tolerance on the part of authors. Arrangements have been concluded for enlarging the text section of our journal next year, remedying somewhat the present lag in publication time.

The details of our new publication arrangement will be presented next month by Gradie R. Rowntree, M.D., in his regular President's Message. The present issue is the last to be published in Chicago. The breaking of a close working relationship with Mr. Clark D. Bridges and Miss Doris L. Flournoy and their associates on our original publication staff is an unpleasant payment for the advantages that will come with the new arrangement. Our final editorial handshake this year is for them, as a very small recognition, indeed, for what they have done.

# Book Reviews

## PSYCHOPHYSIOLOGICAL ASPECTS OF SPACE FLIGHT. Edited by Bernard E. Flaherty. Price, $10.00. Pp. 393, with 52 illustrations. Columbia University Press, 2960 Broadway, New York 27, N.Y., 1961.

By C. CRAIG WRIGHT, M.D.

The space enthusiast will find some interesting articles in this book. It contains 28 papers presented during a symposium of the same title, sponsored by the U.S. Air Force School of Aviation Medicine, in May of 1960. The symposium was called to survey the opinions of informed physicians and scientists concerning present knowledge of man's behavioral capabilities in space and to recommend paths for future research.

Of rather general interest are two papers on Project Mercury—one on its program and objectives, the other on the selection and training of astronauts. Two other papers, on sensory overloading and sensory deprivation, discuss subjects of possible direct application in industrial settings. Every physician will find the paper by Nathan S. Kline, M.D., and Manfred Clynes, "Drugs, Space and Cybernetics: Evolution to Cyborgs," to be stimulating reading.

As implied by the title, all the papers presented have some relation to stresses upon man in space and his physiological reactions as influenced by psychological factors. The term "psychophysiological" is never really defined, allowing the authors to range from psychosocial problems of small groups, through discussions of selection, training, and maintenance, to technical papers on neurophysiology and endocrinology. Several of the papers included touch tangentially on the book title subject, only to ricochet into the author's special field of interest. Written as they were by many authors, the quality of the papers ranges from mediocre to high, with a few outstanding in organization and content.

The collection is inconstant in the degree of technical background required to follow the presentations. Some papers are straightforward and easily understood, while others are filled with jargon terms, sacred to the field and confusing to the uninitiated. Many of the articles presuppose detailed knowledge of the problems of space flight. The casual reader will find that a background of clinical medicine, and particularly physiology, is necessary to appreciate some of the points discussed.

Unfortunately, a few of the authors, selected as recognized experts in their fields, chose to write in the dry, dull, and stilted style becoming known as "American scientific prose," complete with parenthesized numbers in the text and reference listings running to two pages of print. This approach tends to make potentially fascinating subject material a chore to read. In format and writing style, this is not a book for popular consumption, nor is it, by any criterion, a reference book. The physician engaged in industrial medical practice will find little in the book directly applicable to his field of endeavor. To the specialist in aerospace medicine, familiar with the specialty journals, much of the material will seem a rehash of articles previously published. Since there is little interdependence of one paper on another, the reader is encouraged to consult the table of contents for selected readings.

Dr. Wright is Corporation Flight Surgeon for U.S. Steel Corporation, Pittsburgh, Pa.

## SELECTIVE TOXICITY. Adrien Albert, D.Sc., Ph.D., F.R.I.C. Ed. 2. Price, $5.50. Pp. 233, with illustrations. John Wiley & Sons, Inc. 440 Fourth Ave., New York 16, N.Y., 1960.

By E. V. HENSON, M.D.

In this expanded and updated edition, the author devotes approximately 40% of the volume to areas of general interest and information. The basis, application, and accomplishments of selective toxicity, also called comparative biochemistry, are explored, and an adequate explanation is made of the influence which types of membranes have on the permeability of exogenous agents involved in the physiological processes of absorption, distribution, and excretion. Changes which transform inactive or weakly active agents into very active biological materials are exemplified by structural formulas, and several therapeutic, insecticidal, and fungicidal chemicals are used as examples of Paul Ehrlich's definition of chemotherapy—"the injury of an invading organism without injury to the host."

In "Part Two: The Relationship Between Structure and Biological Activity," the author elaborates more specifically on the basic principles in considering such topics as metabolites, enzymes, ionization, surface chemistry, and steric factors, to name a few. In this section also, tables of summarized information, graphs, and structural formulas are used to great advantage in presenting ideas and making comparisons which assist the reader over rough spots which otherwise might be difficult.

Although the book is devoted primarily to selective toxicity as denoted in the title, nonselective depressants that are much less sensitive to small changes in structure are arequately considered in the discussion of "Biological Activity Unrelated to Structure." The straight-line, logarithmic relationship of toxic concentration and solubility of members of a homologous series as shown by Ferguson's Principle has several applications in studying toxic materials, for it may be quickly determined if the mode of action is physical or chemical and appropriate studies can therefore be planned.

Readers with special interests in clinical medicine, biochemistry, pharmacy, therapeutics, and ap-

Dr. Henson is the author of several review articles on the toxicology of organic chemicals. He is an Assistant Editor of this journal, in charge of the abstracts section.

plications of the so-called economic poisons will find something of interest and value in one or more of the thirteen chapters in the two parts. With the aid of well-chosen illustrations and in precise language, the author has covered a difficult subject well.

## MEDICAL FORMS AND PROCEDURES FOR INDUSTRY. Herbert L. Herschensohn, M.D., Price, $8.50. Pp. 192. Charles C Thomas, Publisher, 301 E. Lawrence Ave., Springfield, Ill., 1961.

By JOSEPH RUFFIN, M.D.

After adjusting to the peculiar ordering of the words comprising the title of Dr. Herschensohn's book—logically, procedures antecede their forms—it becomes apparent that there is much within this little volume to interest a variety of industrial medical practitioners.

Dr. Herschensohn has prepared a guide for dealing with the usual gamut of problems encountered by industrial physicians. Surprising as it may seem, this simple book dealing comprehensively with basic policy and procedural matters is unique. The topics given consideration range from "Absence Due to Illness and Injury, Guide to Measurement" to "X-Rays, Routine Views." Between these extremes of the alphabetical listing we observe references to "Dental Injury, Statement of Satisfaction," "Expenses, Outside Medical," "Medical Department, Organization," "Patients, Laid Off," and "Visitors, Injury to" among the approximately 150 different topic headings. The author claims neither originality nor infallibility, readily admitting that the procedures described are a synthesis of his own experience and that of many others.

This manual permits the critically inclined industrial physician to observe his own modus operandi more closely and then to study the comparative anatomy of policies, procedures, and their paper progeny from at least one other source. The result of such an effort should be constructive, not because Dr. Herschensohn has necessarily given us something extraordinary, but primarily because he has provided us with *the opportunity to make comparison*, an exercise which is always potentially fruitful. A commendable feature is the concise and direct presentation of "grey-zone" topics and topics too often discussed with real or feigned delicacy: e.g., sobriety testing; the "special treatment" of top executives; athletic, recreational and parking lot injuries; deaths on company property.

I can imagine some of the sophisticates of occupational medicine laying this book aside with a touch of scorn—"too elementary"—and they are probably right if my touring memories of proudly displayed but reluctantly disclosed policy and procedural manuals permit such a conclusion. Conceding that there are many who need or want no help from Dr. Herschensohn, it should not be forgotten that there are numerous physicians having a casual or a part-time relationship with industry who can make excellent use of this little volume. For these physicians the book should have a high utility quotient. The functional format increases the book's chances of being read, or at least consulted, if not by the visiting doctor, possibly by the nurse, or a manager within the organization who appreciates the fact that the day-to-day functions of a medical service within an industry require a thoughtful and systematic approach as much as any other organizational function. The book's simplicity, both in mode of presentation (alphabetical listing of topics) and conciseness of discussion, is an unqualified asset which should be recognized by all propagandists of the better practice of occupational medicine. I recall too vividly during my public occupational health experience the nearly universal deficiency of satisfactory policy and procedural guides in plants of all sizes; this book would have been an appropriate remedy. Any novice physician initiating an occupational health program will find this manual a reassuring guide. One would hope, of course, that it would serve as the springboard of an original manual taking full cognizance of local needs, customs, and laws.

Dr. Herschensohn says much that provides a basis for argument and I (and probably Dr. Herschensohn) would certainly be disappointed in the industrial physician who was not stirred to offer some form of critique of the author's treatment of various topics; e.g., I question relying on the company's legal department to resolve properly certain questions of medical "ethics" (p. 109) or the inference that a physician can "clinically" make a satisfactory decision as to the capacity of an employee who has been drinking to drive his car "safely" (p. 89).

In summary, Dr. Herschensohn has written an ultrapragmatic manual, and, while it is susceptible to both captious and serious criticism, it has considerable value. Dr. Herschensohn is indeed generous in offering (in the preface) personally to assist inquirers in the development of procedures and forms which are not presented in his book, but I doubt if this "service" is in any way contingent upon purchase of the book. Even if it were, I do not believe that it would provide an adequate rationale for the pricing ($8.50) of this manual. Is it that the publisher feels that most of the sales will be made to affluent and insentient organizations rather than to discriminating and not-so-affluent individuals?

Dr. Ruffin is staff Physician in the Medical Department of the Kaiser Steel Corporation, Fontana, Calif.

## MEDICAL ALMANAC 1961-62. Peter S. Nagan. Price, $5.00. Pp. 528. W. B. Saunders Co., 218 W. Washington Sq., Philadelphia 5, Pa., 1961.

By CHARLES E. LEWIS, M.D.

It would appear that the byproducts of superspecialization are not limited to the field of medicine. The appearance of the first Medical Almanac compiled by Peter S. Nagan should be of some comfort to those persons who enjoy thumbing through

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almanacs, who are "compulsive readers," or who are looking for pearls to dispense to their colleagues or students on hospital rounds. Its major value should be to persons who need a ready source for a wide variety of medical information. This little book has several sections which are devoted to lists of names and addresses of organizations and officials in medicine, institutions that are concerned with education and research in medicine, includes the various medical specialty boards and agencies of medical care of the federal government, vital statistics in medicine, medical manpower statistics, figures on the costs of illnesses, and a gigantic collection of miscellaneous tidbits of information.

All of the sections have been compiled with care. In the vital statistics section, there is much information available, such as a breakdown of the number of full- and part-time physicians in occupational medicine by state. There is, however, no mention of state laws with respect to self-insurance for workmen's compensation. The costs of illnesses section contains a wealth of information regarding various expenditures for medical care in the United States. There is also a section (which may be of considerable practical importance) concerning rules and laws relating to income taxes. There is also a portion of one section devoted to famous men in medicine as well as a list of all congressional representatives and senators. The usefulness of the latter list is perhaps most apparent to individual physicians and needs no further comment here.

In general, the book is deserving of the term "almanac" and should be a valuable reference not only for those interested in occupational health but for physicians in general. The major defect in this volume is a rather inadequate index, which occupies only six pages.

## TOXICOLOGY AND BIOCHEMISTRY OF AROMATIC HYDROCARBONS. Horace W. Gerarde, M.D., Ph.D. Price, not given. Pp. 329, with 89 illustrations. Elsevier Publishing Company (distributed in the United States by D. Van Nostrand Company, Limited, 120 Alexander St., Princeton, N.J.), 1960.

By CHARLES E. LEWIS, M.D.

This small volume is distinctive in two ways: first, the modesty with which the author has reported the work done in this field, much of which is his own, and, second, the fact that there is a tremendous amount of information on the aromatic hydrocarbons within the pages of this little book.

The book consists of two major portions. In Part I there are general considerations regarding the classification, sources, uses, physical properties, and methods of analysis of aromatic hydrocarbons. There is a general discussion of the biochemistry and the toxicological effects of the various members of this family. In Part II there is a discussion of the individual aromatic hydrocarbons of industrial importance. This includes benzene, toluene, and many other monoalkyl and dialkyl derivatives of benzene. There are also separate chapters on dicyclic, tricyclic, and polycyclic aromatic hydrocarbons. Each of the sections on the various specific compounds contains much useful information. As could be expected, there is much repetition from chapter to chapter in the section of prevention, detection, and treatment of exposure. This becomes rather monotonous reading, but it is undoubtedly necessary to repeat the general axioms for each compound for those persons who will use this as a reference text.

There are very few errors in this book, mostly of minor omission rather than commission. For example, on page 229, while discussing the hemolytic anemia which is produced by naphthalene, the author refers to "an abnormality of glutathione metabolism." The problem in such persons is not abnormal glutathione per se, but a deficit of glucose-6-phosphate dehydrogenase, which predisposes to hemolysis of older red cells.

There is a superb final chapter on aromatic hydrocarbon mixtures: solvents, fuels, and lubricants. This should go a long way toward resolving the quandary in many physicians' minds as to the difference between kerosene, gasoline, diesel oil, and other fractions. For those who are not already familiar with the terms used in the petrochemical industry, there is a superb appendix. This is a book in which the average reader might well read the last chapter before beginning the book.

The volume is well printed; it is regrettable that in the review copy there is no statement as to the price and that there is apparently no opportunity of obtaining a more substantial (hardback) copy, since this volume is so full of good information that it should probably be worn thin from constant use by physicians who practice industrial medicine.

## INTRA-OSSEOUS VENOGRAPHY. Robert A. Schobinger, M.D. Price, $14.50. Pp. 243, with illustrations. Grune & Stratton, Inc. 381 Fourth Ave., New York 16, N.Y., 1960.

By HENRY P. PENDERGRASS, M.D.

As stated by the author in his preface, this book was conceived as an atlas "to provide a guide for the clinical interpretation of venograms obtained by intra-osseous route." Normal extraosseous anatomic patterns (along with normal variations) of both deep and superficial venous pathways are demonstrated by roentgenograms and line drawings from the author's experience of some 1,200 injections into various bony sites. Direct injection of contrast material into intra abdominal veins during laparotomy, percutaneous splenopostography, and indirect venography via arterial injection are other means of opacifying the deep venous territories. The techniques of intraosseous approach along with extensive anatomical diagrams are presented lucidly and will serve future investigators well as they attempt to evaluate the clinical application of this procedure.

The bibliography, although not keyed to the text, appears to be complete and up to date.

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Dr. Pendergrass is an associate in the Department of Radiology, Hospital of the University of Pennsylvania, Philadelphia.

Some portions of the venous system are extensively covered with text and illustrations, other areas less well. While at least 15 distinct osseous injection sites are described, the author does not break down his 1,200 injections by site and does not indicate how many of the 1,200 injections demonstrated pathological findings.

This monograph with 317 excellent illustrations and accompanying text will prove to be an invaluable reference to anyone interested in the further evaluation of this diagnostic tool.

**THE MEDICAL DEPARTMENT OF THE UNITED STATES ARMY, SURGERY IN WORLD WAR II: THE PHYSIOLOGIC EFFECTS OF WOUNDS. The Board for the Study of the Severely Wounded, North African-Mediterranean Theatre of Operations. Edited by Henry K. Beecher. Price, $3.50. Pp. 376, with illustrations. Office of the Surgeon General, Department of the Army, Washington, D.C., 1952. For sale by the Superintendent of Documents, U.S. Government Printing Office, Washington 25, D.C.**

By ROBERT C. COMBS, M.D.

This is a most intriguing and stimulating volume prepared by competent investigators and to the best of our knowledge is the only such work in existence. On Sept. 3, 1944, the Commanding General, North African Theater of Operations, appointed a medical board to study the treatment of the severely wounded. Originally primarily interested in the question of anuria in the severely wounded, the investigators found they had collected the material for a physiological atlas of the severely wounded. Their data were accumulated during the rigors of field conditions, at times under fire. In spite of the many obvious difficulties attending their work, these authors were able in a controlled scientific way to study the physiological effects of wounding on a very large number of casualties.

A quick glance at the chapter headings shows the fascinating contents of the book: I. Internal State of Severely Wounded Men on Entry to the Most Forward Hospitals. II. Liver Function in the Severely Wounded. III. Renal Function in the Severely Wounded. IV. "Shock Kidney." V. Clinical, Physiologic, and Biochemic Correlation in Lower Nephron Nephrosis. VII. Effect of Alkalies in Treatment of Traumatic Shock. VIII. Pigment Mobilitation in Severely Wounded Men. IX. Pathology of the Kidney in Traumatic Shock. X. Sulfonamides in Relation to "Shock Kidney." XI. The Crush Syndrome in Battle Casualties. XII. General Pathology of Traumatic Shock.

Though their work usually substantiated the then current understandings of the altered physiology of the acutely injured, of perhaps greater interest were the numerous instances in which their scientific observations discredited long-held clinical concepts.

Dr. Combs, a member of the Editorial Board of this journal, is in the practice of surgery in San Francisco, and an assistant clinical professor of surgery at the University of California School of Medicine.

**SILICOSIS AND OTHER PNEUMOCONIOSES IN SWEDEN. Axel K. J. Ahlmark, M.D. N. E. Torsten Bruce, M.D. and Ake E. Nystrom, M.D. Price, not given. Pp. 399. Svenska Bokforlaget, Stockholm, Sweden, 1960.**

By FRANK PRINCI, M.D.

This volume represents the results of the study of the incidence of industrial pulmonary diseases in Sweden since 1931 in the case of silicosis and since 1950 in the case of the other pneumoconioses. The data were obtained by the authors from government and private insurance carriers and have been arranged to reveal the incidence of pulmonary fibrosis under the conditions of exposure that have existed in various Swedish industries.

The authors have employed the roentgenographic classification of pneumoconioses that was adopted at Johannesburg in 1930. This classification will be more familiar and easier to interpret than some of the more recent classifications. It may lack the specific identifications that are possible with the newer designations but it has the merit of simplicity and universal understanding.

The various chapters are arranged according to specific industries and the industrial exposures are described. In addition, each chapter is presented in the same format so that comparisons among tables in the various sections are easily compared and understood. The general plan of reporting has been explained carefully and clearly in the first part of the book and should be read carefully in order to understand the contents of the volume completely.

This should prove to be a useful reference work and will be of interest to all those who are interested in the pneumoconioses. The translation is somewhat stilted but understandable.

Dr. Princi is Associate Director of the Kettering Laboratory and Professor of Industrial Medicine at the University of Cincinnati.

# Publications Available

*The following list of publications has been compiled on the basis of probable interest to the readers of this Journal and is presented solely for the purpose of giving notice of their availability.*

**Nursing Home Administration.** John D. Gerletti, Ed.D.; C. C. Crawford, Ph.D.; and Donovan J. Perkins, M.S. Price, $6.50. Pp. 472, with 11 illustrations. Attending Staff Assocation, 7601 E. Imperial Highway, Downey, Calif., 1961.

This volume is primarily concerned with training materials for the administration of nursing, boarding, and mental hygiene homes for the aged. It was prepared under a grant from the Bureau of Hospitals, California State Department of Public Health, for the purpose of raising standards of patient care in nursing homes and related institu-

tions through the training of administrators. The table of contents includes chapters on how to care for patients or guests, how to work with families or relatives, public relations, plants and housekeeping, records and recording, money and finances, purchasing, nutrition and personnel relations.

**Leading Diagnoses and Reasons for Patient Visits:** A quarterly report compiled by the National Disease and Therapeutic Index Service of Lea Associates, available from the NDTI Medical Director, 1526 Bethlehem Pike, Flowertown, Pa.

This is a quarterly report in a continuing series of patients seen in private medical practice in the United States. A panel of 1,200 physicans report on all private patient contacts during a 48-hour period once each quarter of a year. The report consists of statistics which break down the causes for patient visits by quarter and by year. The breakdown is on the basis of diseases involving certain organ systems or certain medical and surgical areas of special interest.

# Letters

TO THE EDITOR: Some two years ago your Journal published an article by me (Group Disability Insurance—Abuse and Control, J. OCCUP. MED. 1:451, 1959) that dealt with control of group disability insurance costs. A plan was described that uses two control measures: (a) separation of the Employee's Claim Form from the Physician's Report, the latter thus being removed from the claimant's scrutiny, and (b) impartial examinations in questionable cases by a committee whose membership rotates among members of the local medical society. The numerous requests for reprints of the article suggest a wide interest in the program described. This note is to bring the original report up to date.

The control program was instituted after the second full year of operation of the group disability plan. In that year the benefits paid out so far exceeded the original premium paid that the employer was required to pay an additional 21% of the premium. After the control program was instituted, the claims experience improved to the point that the carrier returned about 20% of the annual premium. Over the eight years of operation of the control program the premium return has averaged 25%. If one adds the premium penalty of the second year to the average premium returns of ensuing years, the control program may be credited with an average saving of 46% of the annual premium of the group disability insurance plan.

The annual experience was as follows:

| Year | Premium Adjustments In Percentages* |
|---|---|
| 1950-51 | — 2.2 |
| 1951-52 | + 21.1 |
| 1952-53 | — 20.6 |
| 1953-54 | — 14.3 |
| 1954-55 | — 21.0 |
| 1955-56 | — 23.2 |
| 1956-57 | — 31.2 |
| 1957-58 | — 23.5 |
| 1958-59 | — 33.5 |
| 1959-60 | — 30.9 |

*— = Premium Refund
+ = Additional Premium

You will observe that the experience of the first two years was characteristic of that of most companies. A modest premium refund the first year was followed by a considerable extra charge the second as employees learned to abuse the plan. The figures for the last two years would have been more impressive but for the fact that a considerable number of our older and impecunious employees who became ill shortly before retirement were permitted to exhaust their full insurance benefits of 26 weeks before retirement became effective. We have had occasion to resort to the impartial examination only five times during the past year.

—ROBERT W. BUTLER, M.D.
Scovill Manufacturing Co.,
Waterbury, Conn.

## IMPORTANT NOTICE

### To Members of The Industrial Medical Association

At the annual meeting of the membership last April the proposed By-laws changes, announced in the March, 1961, issue of the *Journal of Occupational Medicine*, were approved by unanimous vote. These included two major changes:

(1) The annual dues for members was increased to $30.00, and for Fellows, to $35.00, effective January 1, 1962. This action was taken to meet increasing costs of administration and to expand and improve services to the membership.

(2) Officers and directors of the Association will be elected in the future by mail vote and a ballot will be sent each member in good standing in February, 1962. Candidates for officers and directors will be announced in the February issue of the *Journal of Occupational Medicine*.

# THE PRESIDENT'S MESSAGE

GRADIE R. ROWNTREE, M.D.

THE SUBSTANTIAL role which the component societies of the Industrial Medical Association can play and should play in our total program is not, I think, always fully appreciated by our membership. These societies, designed to promote the cause of occupational medicine —its responsibilities, the advancement of its knowledge and skills, and the improvement of its standards—at the local level, can be a strong mortar binding together the masonry of our national structure. We have 19 such component societies at present. I believe our organization even now could support 30.

It is relatively easy, of course, to establish a component society in an area with a large concentration of industries which have organized medical departments. The first ones were founded in such places. In states with heavy groupings of industry, local or county component societies under the state component might well be organized. The recognition of the need for such components is the essential factor.

As you know, the Board of Directors of IMA may establish such a society upon application by 25 or more member physicians, or those having acceptable qualifications for membership, in a given area. Required, of course, is the election of a president, vice president, secretary, treasurer, and a board of directors at an annual meeting. The time of this and other meetings, and if possible the programs planned, should be published in the JOURNAL OF OCCUPATIONAL MEDICINE for the information of all IMA members.

The components provide opportunity for attendance at meetings nearer home and allow for greater participation in programs. If they did nothing more, thus, than afford that professional exchange of ideas with colleagues in the same field that is so vital to all practice, and offer members a more personal chance to learn of new advances in occupational medicine, they would prove their value. But these societies serve also as contacts between members in the region and the national organization. They solicit additional qualified IMA members and approve all applications for national membership in the areas they cover. And they are an authentic local source of professional information and guidance.

The component society will try to have its annual meeting coincide with that of the state medical society, seek to set up programs for county medical and local specialty group meetings, and cooperate in providing speakers in the occupational specialty whenever the opportunity arises. As the local organization representing the Industrial Medical Association in its own region, it maintains liaison with the IMA officer responsible for component societies and constantly works to stimulate interest in the setting up of occupational health programs in its area.

Obviously such a local group is eminently equipped to do something about the most pressing demand in the industrial health field today, namely, meeting the occupational health needs of small business establishments. The component society can encourage physicians in private practice to enter this challenging field on a part-time basis and can help them to expand their medical knowledge and potential for service there.

During this year two new component societies—the Carolina Industrial Medical Association and the Puerto Rico Industrial Medical Association—have been founded. May other qualified groups soon follow!

# Abstracts of Current Literature

## Noise and Hearing

**Békésy Audiometry in Analysis of Auditory Disorders.** J. F. Jerger.* J. Speech & Hearing Res. 3:275 (Sept.) 1960. (*Northwestern University, Evanston, Ill.)

This paper is concerned with Békésy audiometry as a clinical tool in the evaluation of the hearing impaired. The author emphasized that the paper is not concerned with how Békésy audiometry relates with loudness recruitment, only with how it relates with site of lesion within the auditory system. The report is based on the Békésy audiograms of 434 subjects tested at the hearing clinic over a three-year period. Data on one ear of each subject was used. A complete test consisted of two separate tracings on the same piece of graph paper. In one, the signal was periodically interrupted (2.5 ips); in the second it was continuous in time. Analyses of the results indicate a unique relationship between continuous and interrupted tracings which correspond to site of lesion within the auditory system. Most of the tracings could be placed into one of four categories based on the above relationship. Lesions of the middle ear are characterized by one relationship, lesions of the cochlea by another, and lesions of the eighth nerve by a third and fourth relationship. —R.G.H.

**The Indiscriminate Use of Masking in Bone-Conduction Audiometry.** J. F. Jerger* and M. Wertz. A.M.A. Arch. Otolaryng. 70:419 (Oct.) 1959. (*Northwestern University, Evanston, Ill.)

This article emphasizes the need for further research on the problem of a completely satisfactory method for bone-conduction audiometry. The author points out that many authorities contend that the opposite ear should always be masked during bone-conduction audiometry in order to eliminate the possibility that the bone-conducted sound, crossing the skull, will be heard in the nontest ear. Others feel that masking should be used only if the patient actually refers the test tone to the nontest ear. Still other investigators are concerned over the largely unexplored problem of how a rather loud noise in one ear may affect threshold responses in the other ear of clinical patients, irrespective of the cross-hearing problem, and they point out that the noise inevitably activates not only the desired cochlea but the entire complex of higher auditory pathways as well. The author then reports a case history involving a moderate unilateral loss which illustrates how the use of masking on the nontest ear yielded a completely erroneous bone audiogram. Tuning fork tests by four separate otologists indicated a sensorineural, or perceptive, loss in the right ear. With use of a clinical audiometer, the unmasked bone curve when compared to the masked curve had the earmarks of a "shadow" response from the left ear. All other tests on the right ear, however, pointed toward a conductive loss. Low SISI scores, negative Békésy response, and alternate loudness balance tests showed no recruitment, and speech discrimination was excellent. Surgical exploration for stapes mobilization showed the footplate to be completely fixed by otosclerosis. Palpation and percussion of the footplate failed to mobilize, and the ear was closed without improvement of hearing. There was no apparent explanation derived from the surgical procedure which would explain the preoperative discrepancies obtained by audiometry and tuning fork. The author states that the purpose of this report is to stress that as the clinician continues to face situations in which the use of a contralateral masking noise is indicated he should do so with extreme caution and with full awareness of the limitations inherent in the technique. —R.G.H.

## Clinical Toxicology

**The Role of Endotoxins in the Pathogenesis of the Diseases Caused by the Inhalation of Vegetable Dusts.** B. Pernis,* E. C. Vigliani, and G. Cavagna. Med. Lavoro 51:780 (Dec.) 1960. (*Clinica del Lavoro, Milan, Italy)

The clinical similarity which is presented by a variety of respiratory disorders, all caused by one or another type of vegetable dust, led these authors to hypothesize a common causative mechanism.

In the opinion of these authors, since the constituents of these dusts are probably entirely different, the common cause responsible for the respiratory effects and for the fever are more likely bacterial or mold endotoxins. As a matter of fact, Gram-negative bacteria and molds are constant companions of vegetable fibers. To prove their hypothesis, Pernis and co-workers have assembled an extensive series of experimental parallels, some derived from careful literature research, between biological effects of bacterial endotoxins and those of cotton dust extracts. These parallels point rather strikingly to the similarity of effects of the two groups of agents in relation to various immunological phenomena: skin reaction, leucocytic infiltration, respiratory and blood pressure effects, histamine liberation, etc. In the hands of these authors endotoxins given by inhalation to rabbits manifested conspicuous respiratory effects, similar to those of the inhalation of cotton dust. The phenomenon of tolerance, or reduced response, in consecutive exposures, appears evident with endotoxins given by aerosol both in terms of fever induction and in respiratory distress. Human subjects exposed to aerosols of Escherichia coli endotoxins, presented acute respiratory effects similar to those recorded with cotton dust inhalation. In one case,

the response was quite dramatic, characterized by protracted and severe respiratory and general distress. Extrapolating from these observations, the authors felt that they were justified in considering that a common etiology existed for the entire group of respiratory disturbances caused by vegetable dusts (mill fever, mattress-maker's fever, hemp fever, heckling fever, farmer's lung, printer's disease, etc.). Gram-negative bacterial endotoxins, which act through the liberation of histamine from the mast-cells, are believed to be the most probable factor. —M.C.B.

## Experimental Toxicology

**The Influence of Pore Size on Carcinogenicity of Subcutaneously Implanted Millipore Filters.** P. Goldhaber.* Am. A. Cancer Res. Proc. 3:228 (March) 1961. (*Harvard School of Dental Medicine, Boston, Mass.)

During the course of experiments with Millipore diffusion chambers containing various tissues, it was noted that large fibrosarcomas occasionally developed around chambers implanted subcutaneously for periods of longer than six months in Swiss albino mice. Although the exposed edge of the Plexiglas ring could account for the entire carcinogenic effect of the chamber, it was decided to test Millipore filters (19 mm diameter and 150 $\mu$ thick) of various pore sizes as well. One year following subcutaneous implantation of these materials, it was noted that, whereas the Plexiglas rings (hole in center) have rise to fibrosarcomas in 11% of the animals (4/36), the incidence around Millipore filters appears to be inversely to pore size. Only 1/39 developed a tumor around the 450 $\mu$ pore-size filters 9/34 around the 100 $\mu$ filter, and 16/35 around the 50 $\mu$ size. The potency of certain physical carcinogens may thus be directly related to their degree of interference with normal diffusion rates of specific nutrients or metabolites to or from the surrounding cells. —R.E.E.

**Acute and Subacute Toxicity of Dimethoate.** B. West,* L. B. Vidone, and C. B. Shaffer. Toxicol. & Appl. Pharmacol. 3:210 (March) 1961. (*American Cyanamid Co., Stamford, Conn.)

The study was designed to determine the minimum dietary level of dimethoate which is effective in depressing plasma and erythrocyte cholinesterase and the maximum dietary level which permits survival for several months. The $LD_{50}$ was found to be 185-215 mg/kg for male rats and 245 mg/kg for females; the susceptibility difference between the sexes is not considered significant. Acut oral toxicity studies on rats with mixtures of dimethoate and the organic phosphates that had already been granted pesticide residue tolerances revealed that no greater-than-additive toxic effect occurred with any pair of compounds. Dimethoate is not readily absorbed through the skin and is not irritating to the skin or eyes. Erythrocyte cholinesterase is more readily inhibited by dimethoate than is plasma cholinesterase. The minimally effective 90-day dietary level for rat plasma and erythrocyte inhibition lies between 32 and 50 ppm; in dogs erythrocyte cholinesterase is depressed slightly at 50 ppm but plasma cholinesterase is unaffected. Relatively high levels fed to rats and dogs for 90 days markedly depressed cholinesterase activities but did not cause signifificant hematologic changes or anatomical pathology. —R.E.E.

## Medical Care Administration

**Medical Students, Family Doctors, and Family Practice.** T. E. Rardin.* J.A.M.A. 176:479 (May 13) 1961. (*Section on General Practice, AMA, 1975 Guilford Road, Columbus, Ohio)

Everyone seems to agree that there is no substitute for a personal or family doctor: yet each year fewer medical school graduates become family physicians. Contrary to the reaction of the Advisory Council on Medical Specialties of the American Medical Society, the author maintains that family practice is a specialty and a family doctor a specialist, provided he has been trained for, develops, and demonstrates skill in relating his wide general perspective of comprehensive medicine in a single person or a family group over a continuing period of time.

We need more family doctors on medical school faculties, accepted and recognized as equally important in the educational scheme. Student contacts are important in motivating students to select the aspects of medicine they will pursue. All specialty areas except that of the family doctor are represented by live, eager, enthusiastic personalities.

The family doctor often has the temperament, patience, and ability to talk with students, where highly specialized teachers often do not. Our deans and medical school faculties have a great opportunity to develop this long neglected aspect of undergraduate teaching. —A.B.

## Dermatology

**The Pathogenesis of Miliaria.** L. J. A. Loewenthal.* Arch. Dermat. 84:2 (July) 1961. (*South African Institute for Medical Research, Johannesburg, South Africa)

The author presents his view of sweat retention, a common and sometimes difficult problem which has received much attention in the dermatologic literature in recent years, after histological examination of naturally occurring lesions in 21 miners. He confirmed earlier workers' observations of poral closure by a keratotic or parakeratotic mass, dilatation and occasional rupture of the sweat duct, and surrounding inflammation; but he claims these are late findings. He categorically denies that intraluminal periodic acid—

Schiff-positive material plays any role in the pathogenesis of miliaria. The earliest lesion according to Loewenthal consists of localized areas of intercellular and intracellular edema, usually situated paraductally. The location and type of reaction suggest an irritant effect from a substance oozing out of sweat ducts. The author favors salt as the irritant. To support this view, he notes that skin soaked in 8% NaCl solution showed the early changes of sweat retention, namely, spongiosis and vesiculation, about twice as often as did skin sites soaked in salt-free solutions. This, of course, cannot be construed as proof; the histologic changes mentioned occur with many mild irritants. The author, nevertheless, has made a provocative suggestion. Perhaps moderate salt restriction will benefit susceptible persons when they are exposed to intense heat for prolonged periods. —W.L.E.

## Industrial Hygiene Controls

**Snowscape Eye Protection.** E. E. Hedblom.* Arch. Environ. Health 2:685 (June) 1961. (*Division of Cold Weather Medicine, U. S. Naval Medical School, National Naval Medical Center, Bethesda, Md.)

Snowblindness has been an affliction of Arctic inhabitants since time immemorial. To develop reasonable controls, it ultimately became necessary to reevaluate all existing protective eyewear. A table summarizes the type of eye protection used by each Arctic expedition since 1902 as well as the reported effectiveness of the protection.

Under actual field conditions, 20 types of dark glasses were investigated. Subjects used the goggles and glasses under three different light conditions and were then asked to list the goggles and glasses in order of preference. Basically, this procedure tested the glare factor. Preference was noted for the following, in descending order: neutral grays, glasses and plastic; Ray-Ban and Colobar greens; Polaroids, with gray preferred to green or gray-green; rose-smoke; plastic green lenses; yellow glasses; and plastic yellow shields. Additional prolonged intensive preference testing was undertaken by 12 officers using prescription glasses of various types. Again the neutral-gray glasses headed their preference ratings. It became apparent that the glare factor was not the only criterion for the preference, for, if it were, the order of preference would have been from dark to light as follows: rose-smoke plus Polaroid; neutral grays; green "Plasticor"; "Ray-Ban No. 3" plus nickel; rose-smoke; and Polaroids. A study of light transmission curves indicated that the transmission of infrared rays in the range of 800-1,500 $\mu$ was a factor in producing eye discomfort.

To name the discomfort caused by intensive exposure to visible light or infrared, the author coined the term "calorophthalgia," which infers the etiological effect of heat on the eye. Dark glasses reduce the transmission of visible light but cause a relative pupillary dilation, which, in turn, permits the transmission of more infrared energy to the eye.

To quantitate the relationship of visible to infrared light transmission, "it was arbitrarily decided to divide the average light transmittance inside the visual threshold by the average transmittance in infrared from 680 to 900 $\mu$, the quotient being the calorophthalgic index." Field experiences indicated that a glass must have an index of 0.8 or more to be comfortable on prolonged use.

The author used this index as an additional new tool in developing and adequate protective glass. Spectral transmission studies were undertaken to determine the effect of such variables as type and color of glass, bath of glass, lens thickness, and lens coatings. In adddition, an analysis of spectacle frames was made. The net result was that a neutral-gray glass, coated with double gradient density Inconel and mounted in and aviation-type metal frame was developed which could be ground to prescriptions of + 2.00 to − 3.00 diopters.

The new protective glasses have been used by various service units having Antarctic bases as well as by 400 scientists of the IGY. "After four years of use in the Antarctica by approximately 2,400 Americans, not only has there never been a case of snowblindness contracted while wearing these glasses, but also there has never been a vocal or recorded complaint relative to eye discomfort, strain, fatigue, or related symptoms contracted while wearing these glasses." —L.W.S.

## Radiation Health

**Irradiation and Marrow Infusion in Leukemia.** E. D. Thomas;* E. C. Herman, Jr.; W. B. Greenough III; E. B. Hager; J. H. Cannon; O. D. Sahler; and J. W. Ferrebee. Arch. Int. Med. 107:829 (June) 1961. (*The Mary Imogene Bassett Hospital, Cooperstown, N. Y.)

Five patients with acute leukemia were given treatment by total-body irradiation and marrow infusion. The dose ranged from 1,460 r to 2,016 r, and the rates ranged from 0.5 r to 2 r per minute. Death occurred in all cases in a period ranging from 13 to 72 days. Marrow recovery occurred in two of the cases while partial recovery occurred in one. The cause of death was infection in three cases, jaundice of undetermined etiology in one, and recurrent leukemia in one. —W.D.N.

**Value of Bone Marrow and Spleen Cell Suspensions for Survival of Lethally Irradiated Dogs.** R. D. Sullivan,* G. Stecher, and S. S. Sternberg. J. Nat. Cancer Inst. 23:367 (Sept.) 1959. (*Sloan-Kettering Institute, New York, N. Y.)

Eighty-five mongrel dogs were given supralethal and marginally lethal doses of total body x-radia-

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tion, and the protective effect of homologous marrow and homologous spleen and of autologous marrow and autologous spleen was studied. At supralethal radiation doses of 600 r, 550 r, and 450 r, no protection was achieved. At an $LD_{8s}$ of 400 r, autologous marrow gave the dogs a considerable degree of protection. —W.D.N.

**Dogs That Survive "Lethal" Exposures to Radiation.** E. B. Hager, J. A. Mannick, E. D. Thomas, and J. W. Ferrebee.* Radiat. Res. 14:192 (Feb.) 1961. (*Mary Imogene Bassett Hospital, Cooperstown, N. Y.)

A group of 37 beagle dogs survived exposures of 600 to 1,800 r of gamma or x-ray administered continuously at dose rates of 2 to 18 r per minute. Treatment consisted of blood or bone marrow transfusions, antibiotics, gamma globulin, hyperimmune serum for distemper and hepatitis, pancreatic extracts, and vitamins. Survival occurred with blood transfusions in 3 instances, homologous bone marrow transplantations in 4 instances, and autologous marrow infusions in 30 instances. The latter proved most efficacious with evidence of returning marrow function within four or five days after these infusions. Dogs given homologous marrow usually did poorly and had high mortality from infections. Intestinal damage seemed to limit successful recovery to exposures of 1,800 r or less in dogs.

Survivors appeared to have normal health and behavior 6 to 20 months after exposure, although their coats are prematurely gray. Three survivors have been permitted to breed and have produced normal offspring. The four survivors treated with homologous marrow appear to be chimeras. The segmented polymorphonuclear leukocytes in the peripheral blood in these male beagles now bear female sex characteristics. —G.L.V.

**Measurement of Bone Marrow and Gonadal Dose from the Chest X-Ray Examination as a Function of Field Size, Field Alignment, Tube Kilovoltage and Added Filtration.** E. R. Epp,* H. Weiss, and J. S. Laughlin. Brit. J. Radiol. 34:85 (Feb.) 1961. (*Division of Biophysics, Sloan-Kettering Institute for Cancer Research, New York)

Measurements of bone marrow and gonadal dose were made in a human phantom during conventional posterior, anterior, and lateral chest x-rays under a wide range of conditions. The conditions included different field sizes, small changes in the angle of the beam, kilovoltage ranges from 60 to 120 kvp, with various additions of filtration. While it was important to have proper conditions covering all these parameters, accurate collimation of the useful beam is by far the most important single factor in minimizing the dosage delivered per milliampere-second to the active bone marrow and the reproductive region of patients undergoing the chest x-ray examination. —W.D.N.

**Fallout Radioactivity in Cattle and Its Effects.** M. A. Van Dilla,* G. R. Farmer, and V. R. Bohman. Science 133:1075 (April 7) 1961. (*Los Alamos Scientific Laboratory, University of California, Los Alamos, N. Mex.)

In 1957, the U. S. Atomic Energy Commission authorized a project to determine what effects, if any, the radioactivity resulting from atomic bomb tests at the Nevada Test Site was having on cattle grazing in areas heavily contaminated by fallout as compared with cattle at a distance.

Fission product level in the rumen contents and fecal samples from the Nevada test herd showed a close correlation with Nevada atomic bomb tests. The levels of cesium-137 and strontium-90 in cattle on the test site and elsewhere in Nevada are quite similar to the levels in cattle from other parts of the country. There has been no evidence to date of biological injury either of a gross or histological nature. —W.D.N.

## Internal Medicine

**Recurrent Pericardial Effusion After Nonpenetrating Chest Trauma—Report of 2 Cases Treated with Adrenocortical Steroids.** M. J. Goodkind,* W. E. Bloomer, and A. V. N. Goodyer. New England J. Med. 263:874 (Nov. 3) 1960. (*Yale University School of Medicine, New Haven, Conn.)

Nonpenetrating chest injuries can cause pericardial effusion and cardiac tamponade. Recurrent effusion and tamponade can occur weeks or months after the initial injury.

The authors report 2 cases in detail including electrocardiograms and chest x-rays. In the first case, tamponade did not occur until two months following an automobile accident. Tamponade in the second case followed a chest injury after a two-week interval. Early diagnosis is important, the usual signs and symptoms being orthopnea, distended neck veins, a weak impulse at the apex with distant heart sounds, a diminished $P_2$, and a pulsus paradoxicus. X-ray studies show a changing cardiac contour when body positions are changed.

Treatment is aimed at relieving the acute tamponade, preventing recurrent effusion, and preventing constrictive pericarditis. Pericardiocentesis rapidly and safely relieves the acute tamponade. Pericardiectomy is being used with success in controlling recurrent effusion and also in preventing constrictive pericarditis.

In both the cases reported, steroid therapy was used prior to pericardiectomy with good results as to relief of symptoms and reducing the effusion. —S.H.S.

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# About the Authors

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► ARTHUR M. PHILLIPS, M.D., is the author of the article in this issue, "A Study of Prolonged Absenteeism." This is based on experience at Weirton Steel Company, Wierton, W. Va., where he is Assistant Director of Medical Services.

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► RICHARD D. STEWART, M.D., heads the list of authors of the article in this issue, "Human Exposure to Carbon Tetrachloride Vapor: Relationship of Expired Air Concentration to Exposure and Toxicity." The point is made in the material submitted with the article that there really is no senior author. The article is the result of experimental work conducted through the cooperation of three laboratories within The Dow Chemical Company: the Chemical Physics Research Laboratory, the Biochemical Research Laboratory, and the Medical Department. It is an extension of the cooperative research which is being carried out to assess the effects upon humans of exposure to chlorinated hydrocarbons, the results being the product of interweaving of disciplines increasingly required to understand some of the problems of applied toxicology.

Dr. Stewart received the A.B. degree from the University of Michigan, Ann Arbor, Mich., with distinction in 1951, and the M.D. degree from the same institution in 1955. After a rotating internship


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at Saginaw (Mich.) General Hospital, he took employment as an industrial physician in the medical department of The Dow Chemical Company, Midland, Mich., with some time given to work in the firm's Biochemical Research Laboratory. While the presently reported work was going on, he was a resident in medicine at the University of Michigan Medical Center, Ann Arbor, Mich. ((1959-1962); and in 1961 he received an AEC Fellowship in Industrial Medicine. The other members of this group of authors are:

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J. E. PETERSON who holds the M.S. degree in chemical engineering from the University of Michigan. He began work at The Dow Chemical Company in the fall of 1952 on "Special Project Assignment" and in the fall of 1953 joined the Environmental Laboratory and has been there since. His interests have been in ventilation (especially the evaluation of laboratory hood performance), the effect of thermal stress on workmen, and the health aspects of the preproduction stage of chemical manufacturing.

► ROBERT K. DAVIS, Ph.D., is author of the topical review in this issue, "Fluoride Intoxication in Laboratory Animals." This is the final member of a five-part review which has come from Kettering Laboratory, Cincinnati, Ohio, on the toxicology of fluorides.

Dr. Davis received the A.B. degree, cum laude, from Kenyon College, Gambier, Ohio, in 1937; and the M.A. degree in physiology from Reed College, Portland, Ore., in 1937. He was awarded the Ph.D. degree in physiology by Indiana University in 1948. His doctoral work emphasized endocrine physiology and animal genetics.

Dr. Davis served in the Medical Corps, U.S. Army, in the Philippine Islands and Japan during 1943-1946. In his professional appointments he has been Section Chief, Animal Physiology Section, Biology Division, (U.S.A.E.C.) Mound Laboratory, Miamisburg, Ohio; Toxicologist, Water Pollution Branch, (U.S.P.H.S.) Taft Engineering Center, Cincinnati, Ohio; and Assistant Chief, Physiology Branch, Aeromedical Laboratory, Wright-Patterson Air Force Base, Ohio. He joined the University of Cincinnati (Ohio) in 1957 as Assistant Professor of Industrial Health. —EDITOR

SKIN DISORDERS

RESPONSIVE TO TRIAMCINOLONE

"Triamcinolone has been shown to have more profound anti-inflammatory and antiallergic properties than preceding corticosteroids."*

■ enhanced anti-inflammatory, antipruritic, antiallergic effects ■ less gastrointestinal distress ■ may be of value when other corticoids have failed ■ virtually **no** mood changes, edema, sodium or water retention, or secondary hypertension

**Supply:** Scored tablets of 1 mg., 2 mg. and 4 mg. Syrup in 120 cc. bottles, each 5 cc. teaspoonful containing 5.1 mg. triamcinolone diacetate providing 4 mg. triamcinolone.

*Edelstein, A. J.: Pennsylvania M. J. 62:1831 (Dec.) 1959.

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*KENACORT IS A SQUIBB TRADEMARK.

Pemphigus vulgaris

# People and Events

► HERMAN J. BENNETT, M.D., has been appointed Medical Director of the Wilmington, Del., plant of the Buick-Oldsmobile-Pontiac Assembly Division of General Motors Corporation. Until his present appointment he was Medical Director of the Poughkeepsie, N.Y., plant of the International Business Machines Corporation, and also served on the staff of Vassar Hospital as a consultant in Preventive Medicine and Occupational Medicine.

DR. BENNETT was graduated from New York University with a B.S. degree and received his medical degree from the University of Vienna in 1936. He interned at the Bushwick Hospital in Brooklyn, N. Y., and was a resident at the U.S. Public Health Service Hospital in Lexington, Ky. Following this, he did general practice and part-time industrial medicine and was on the staffs of Lenox Hill and Lincoln Hospitals in New York City.

From 1942 to 1946, DR. BENNETT was on active duty with the Army of the United States. After separation from service, he attended Columbia University and received his M.D.H. degree in 1947. He served as an Associate Physician with the New York State Department of Labor, Workmen's Compensation Board from 1947 to 1956. His major activities were in the fields of occupational diseases and toxicology. During this period, DR. BENNETT was also on the staff of the Adult Cardiac Clinic, Work Classification Unit, Bellevue Hospital. From 1949 to 1958 he was a lecturer in Occupational Medicine in the Department of Preventive Medicine, New York Medical College.

Herman J. Bennett, M.D.

Laurence H. Ballou, M.D.

DR. BENNETT is certified in occupational medicine by the American Board of Preventive Medicine. He is a Fellow of the American College of Preventive Medicine, the American Academy of Occupational Medicine, the Industrial Medical Association, and the American Public Health Association. He is Chairman of the IMA's Emergency and Disaster Planning Committee and a member of the Committee on Problem Drinking.

► LAURENCE H. BALLOU, M.D., has been named Medical Director of The Firestone Tire and Rubber Company, Akron, Ohio. He succeeds WARREN L. HOGUE, M.D., who retired November 1 after 32 years service with the company.

DR. BALLOU has been a staff physician at Firestone for the last four years. Prior to his joining the Company in September, 1957, he was in private practice in Vermont. He was born in Tientsin, China, and attended elementary and secondary schools at Peking American School in Peking, China. His parents were American missionaries in China at the time. He came to the United States in 1939 to enroll at Yale University. After an interruption for military service, he continued his studies at the University of Vermont and was graduated with


**OCCUPATIONAL HEALTH PHYSICIAN**

Certified in Occupational Medicine by the American Board of Preventive Medicine or with acceptable equivalent qualifications including at least three years Occupational Medicine experience.

To work as Occupational Medicine and Workmen's Compensation consultant to Medical Department of Company in the Middle East. Company provides medical care to employees and dependents (300 bed modern hospital, 60 physicians including specialists, 2000 employee admissions and 200,000 employee clinic visits annually).

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a bachelor of arts degree in 1947 and a medical degree in 1951.

DR. BALLOU is a member of the American Medical Association, Industrial Medical Association, and is chairman of the Health and Safety Committee of the Boy Scouts of America.

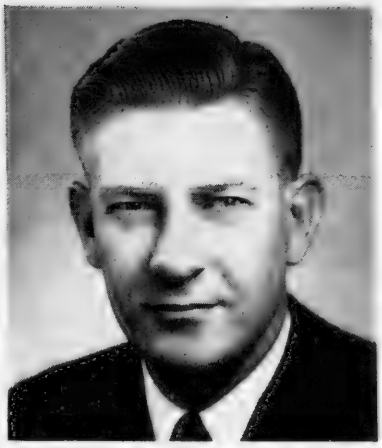
DR. HOGUE joined Firestone in 1922 as Assistant Medical Director and since 1946 has directed the medical and nursing personnel of the company and all of its subsidiaries. He earned both his B.S. degree and his M.D. degree from Northwestern University. He served his internship at Michael Reese Hospital and at Sarah Morris Memorial Children's Hospital, Chicago.

DR. HOGUE is a member of the American Medical Association, the Medical Advisory Commission of the Common Cold Foundation, and a Fellow of the Industrial Medical Association. He is listed in *Who's Who in America* and is serving on the boards for the Summit County Heart Association and the Rheumatism and Arthritis Foundation.

► DON G. FOWLER has been named Health and Safety Director for the Lead Industries Association, New York City, succeeding the late MANFRED BOWDITCH. Before joining Lead Industries, MR. FOWLER was Chief of Industrial Hygiene for the Chrysler Corporation. In his new position he will be responsible for the LIA continuing program for gathering data and assisting in the setting of standards and acceptable practices for the industrial and domestic use of lead and lead products. An important new phase of LIA's industrial hygiene program to be initiated under Mr. Fowler's direction concerns several of the more recent developments in lead technology such as lead and leaded products for the control of radiation hazards and the use of lead in noise and vibration control and abatement.

Mr. Fowler, who is a graduate chemist, has been concerned with safety and industrial hygiene for the past 20 years. Prior to his association with Chrysler, he was regional supervisor for the Michigan Department of Health. Still earlier he was a member of the safety and industrial hygiene staff of the Employers Group Insurance Company.

Among the professional organizations of which Mr. Fowler is a member are the American Society of Safety Engineers, the American Industrial Hygiene Association, the American Association for the Advancement of Science, the Air Pollution Control Association and the Engineering Society of Detroit. He is also a past-president of the Michigan Industrial Hygiene Association and was chairman of the Carbon Monoxide Sub Group of the Automobile Manufacturers' Association.



Don G. Fowler

► ROBERT R. DUGAN, M.D., has been appointed Medical Director of Continental Oil Company, succeeding W. O. ARMSTRONG, M.D., who retired last September after 32 years as Medical Director.

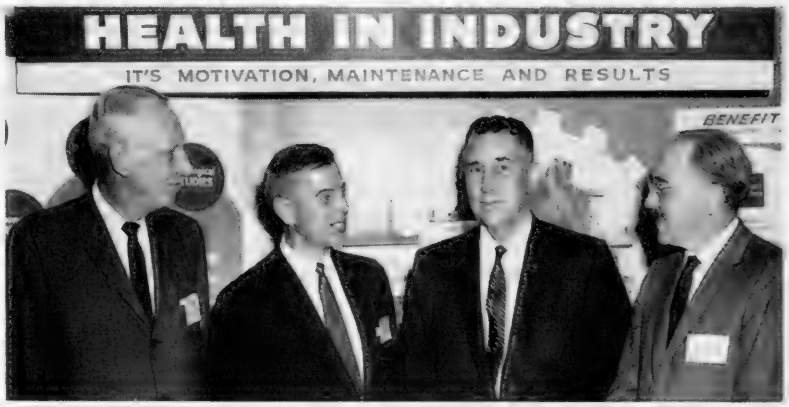
DR. DUGAN has been with Continental Oil since 1952. He joined the Company as Assistant Chief Surgeon and was located in Ponca City, Okla., until a year ago when he was transferred to Houston, Tex., as Assistant Medical Director. He will continue to make his headquarters in Houston.

DR. DUGAN received his M.D. degree from Hahnemann Medical College, Philadelphia, in 1945. He served his internship at Misericordia Hospital in Philadelphia.

► The 26th annual meeting of the Industrial Hygiene Foundation was held in Pittsburgh on October 25 and 26.

H. H. SCHRENK, PH.D., Managing Director of the Foundation, pointed out the public's concern over the environmental aspects of health and well being in his opening remarks at the Management Conference. "More and more attention is being directed to the effects of the environment on the health and well-being of man. The public is concerned about radioactive fall-out, air pollution, food additives, stream pollution and even the exotic hazards of outer space. The publicity given to any one of these areas reflects on the others. A problem of this scope and public interest inevitably brings exaggeration, misinformation and demands for regulations and controls of hazards some of which are nebulous and ill-defined on the present state of knowledge," he said.

That these problems are receiving the attention of research and management groups is evident from the




**Pictured at the 26th annual meeting of the Industrial Hygiene Foundation are (left to right) Dr. Allen D. Brandt, Bethlehem Steel Company; Dr. L. V. Cralley, Aluminum Company of America; Stanley M. Jarrett, Braden Copper Company; and Prof. T. F. Hatch, University of Pittsburgh. The meeting was held in Pittsburgh on October 25 and 26.**

broad scope of papers that were presented at the meeting.

► RICHARD M. ADAMS, M.D., has joined the staff of the Medical Department of Humble Oil & Refining Company, Houston, Tex. Prior to accepting his present position, DR. ADAMS was Medical Director for Interstate Oil Pipe Line Company in Shreveport, La.

DR. ADAMS attended the University of Oklahoma and Southwestern State Teachers College, Weatherford, Okla., and received his medical degree from the University of Arkansas School of Medicine in 1934. He received the master of public health degree from The Johns Hopkins University in 1939. He is a Fellow of the Industrial Medical Association.

► W. CLARK COOPER, M.D., has been named Deputy Chief of the Division of Occupational Health, U.S. Public Health Service. Two other appointments in the Division are DR. AUSTIN HENSCHEL, Chief of the new Physiology Section; and DR. OTTO A. BESSEY, Chief of Research and Training Grants.

DR. COOPER received his medical degree from the University of Virginia in 1934 and continued his studies in internal medicine and nutrition. He later did extensive work on the chemotherapy of malaria at the National Institutes of Health. He joined the Division of Occupational Health in 1952 as Chief of the Occupational Health Field Headquarters in Cincinnati, Ohio. In addition to his new assignment, Dr. Cooper will continue to serve as Chief of Program Planning and Analysis.

DR. COOPER holds the Master of Public Health degree from the Harvard School of Public Health, and

**At the Industrial Hygiene Foundation meeting (left to right): Dr. H. H. Schrenk, Industrial Hygiene Foundation; Dr. Paul C. Cross, Mellon Institute; and Dr. William P. Yant, Mine Safety Appliances Company.**

**At the Industrial Hygiene Foundation meeting (left to right): Dr. John A. Zapp, Jr., E. I. du Pont de Nemours & Company; Dr. Anna M. Baetjer, The Johns Hopkins University; Dr. Paul Gross, Industrial Hygiene Foundation; and Dr. Joseph F. Treon, Atlas Chemical Industries, Inc.**

See you in the
WINDY CITY!
April 10, 11, 12—Chicago
AMERICAN INDUSTRIAL HEALTH CONFERENCE

is certified by the American Board of Internal Medicine. He is a Fellow of the American Association for the Advancement of Science and a member of the American Medical Association, the Industrial Medical Association, the American Public Health Association, and the American Conference of Governmental Industrial Hygienists.

DR. HENSCHEL was formerly Chief of the Environmental Protection Research Division of the Army's Quartermaster Research and Engineering Command. Previously he had served as Research Director of the Quartermaster Climatic Research Laboratory. He was on the faculty of the Physiology Department at the University of Minnesota Medical School and School of Public Health for 12 years.

DR. HENSCHEL received the B.S. and M.A. degrees in zoology and his Ph.D. in physiology from the University of Minnesota. He is a fellow of the American Association for the Advancement of Science and a member of the American Physiological Society, the Society for Experimental Biology and Medicine, the American Association of University Professors, and Sigma Xi.

DR. BESSEY also comes to the Division from the Quartermaster Research and Engineering Command Laboratories of the Army, where he was Associate Chief of the Environmental Protection Research Division. Earlier DR. BESSEY was professor of biochemistry and nutrition at the Universities of Texas and Illinois and was on the staff of the Departments of Pathology and Biochemistry at the Harvard

### Certification

The American Board of Preventive Medicine announces that the next examination for certification in Occupational Medicine will be at the Pick-Congress Hotel, Chicago, April 7-9, 1962.

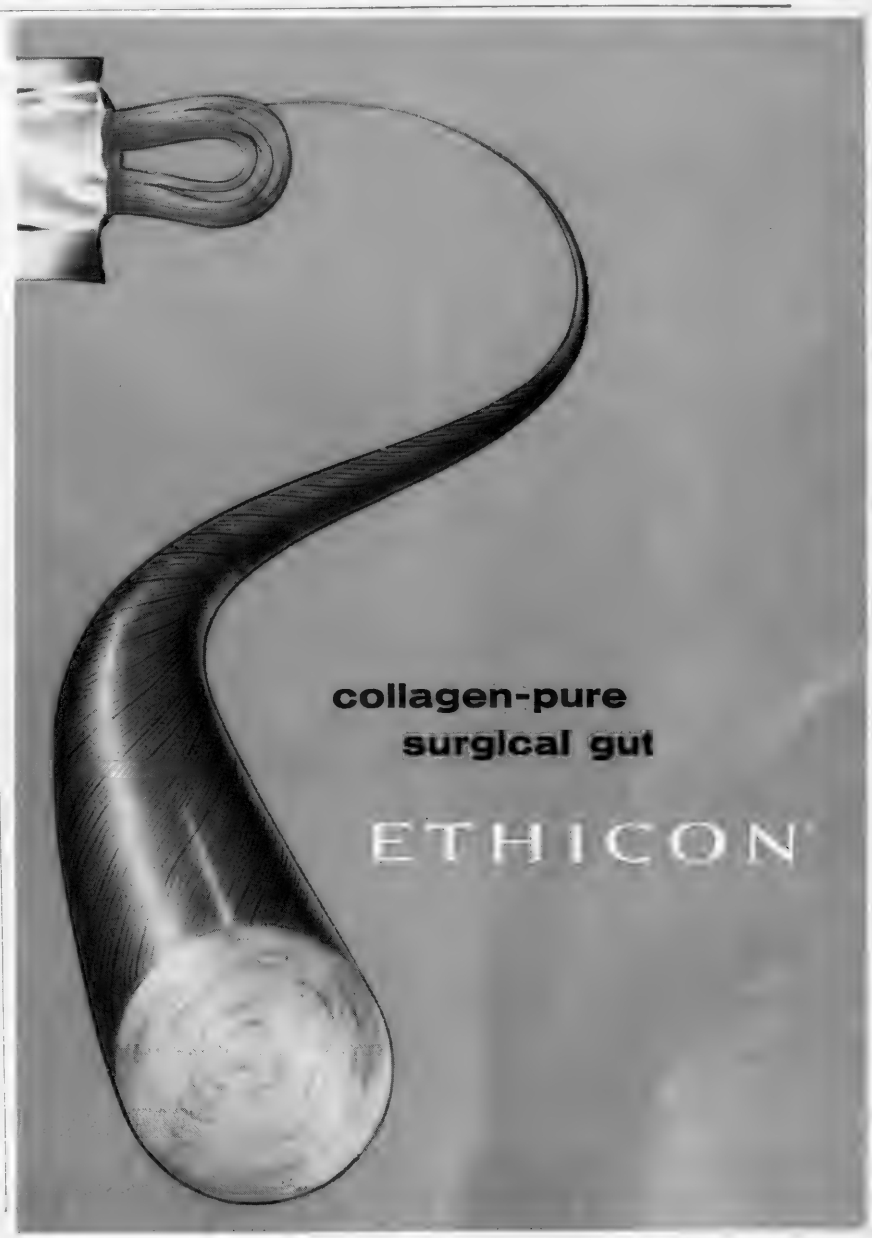
## Death

EMERY R. HAYHURST, M.D., Columbus, Ohio, on October 19 at age 81.

DR. HAYHURST was a long-time servant of public health in Ohio and a major contributor of knowledge in the field of occupational health. He was engaged in industrial medical work for 50 years. He was associated with the Ohio Department of Health from 1913 to 1936. He also taught at Ohio State University and was a consultant on industrial medicine.

He received his M.D. degree from the University of Illinois College of Medicine in 1908. He was one of the founders of the American Association of Industrial Physicians and Surgeons, early name of the Industrial Medical Association. He became a Fellow in 1931 and was made an honorary member in 1951.

Under DR. HAYHURST'S guidance, Ohio became one of the first states to establish an occupational health division in a state health department. Early in this program information was gathered which resulted in the publication in 1915 of a 483-page volume entitled "A Survey of Industrial Health Hazards and Occupational Diseases in Ohio," for many years a basic guide, not only in Ohio, but also in other states.



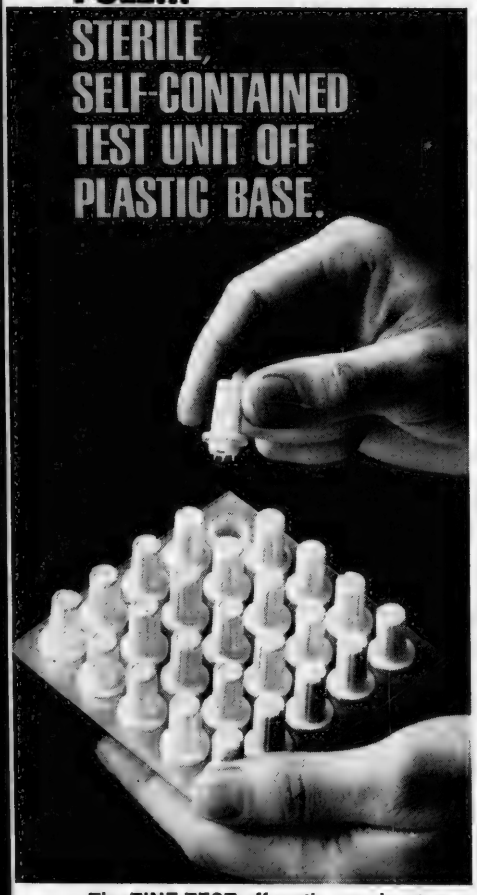
# This is the highly accurate Tuberculin, Tine Test

Each unit contains one complete tuberculin test: a plastic holder with stainless steel disc and 4 tines coated with Old Tuberculin and dried. Except for cleaning the site with alcohol, no preparation, clean-up or other materials are needed. Reactions, read 48-72 hours later, can be readily identified and measured accurately. A series of tests* performed in tuberculosis hospitals confirmed the accuracy of the Tuberculin, Tine Test compared with the Mantoux. In one group of 942 Mantoux positives, 97.8 per cent were positive by the Tine Test. Among another group of 436 bacteriologically proven cases, 97.7 per cent were positive by the Mantoux Test and 97 per cent by the Tine Test. For industrial, school or community programs, the LEDERLE TUBERCULIN, TINE TEST offers distinct advantages in speed, accuracy and economy.

**Supplied:** Box of 25 test units.

*Russell, W. F., Jr.; Lynch, H. J.; Doto, I.; and Furcolow, M. L.; Comparison of Tuberculin Skin Reactions: Tine Test vs. Intracutaneous, Scientific Exhibit, 110th Annual Meeting, A.M.A., New York, June 26-30, 1961.

**PULL...**




**PRESS...**

**DISCARD**

**The TINE TEST offers these advantages:** ▪ Accuracy comparable to the Mantoux ▪ Adaptable to both mass testing and individual use in office practice ▪ Sterile, and disposable after one use—eliminating possible danger of cross-infection ▪ Easy to apply, permits uniform administration by auxiliary personnel ▪ Designed for rapidity, a physician and nurse team can test 25 individuals within two minutes ▪ Predetermined dose not subject to errors of measurement ▪ Multiple punctures help eliminate test failures, permit reactions to be read quantitatively ▪ Virtually painless, not frightening to children.

# NEW LEDERLE TUBERCULIN, TINE TEST

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Request complete information on indications, dosage, precautions and contraindications from your Lederle representative or write to Medical Advisory Department.

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**Announcement**

Applications are being solicited for Scientific Exhibits for the 1962 Industrial Health Conference which will be held at the Pick-Congress Hotel in Chicago. Exhibits will be shown April 10 to 12. Application blanks and information may be obtained from Allen P. Skoog, M.D., G. D. Searle and Company, P.O. Box 5110, Chicago 80, Ill.

Medical School and the Harvard School of Public Health.

DR. BESSEY received his bachelor's degree from Montana State University and his Ph.D. degree from the University of Pittsburgh. He belongs to the American Association of University Professors, the American Chemical Society, the American Institute of Biological Chemists, and Sigma Xi.

► GEORGE R. SHORE, M.D., has been appointed part-time company physician for the H. J. Heinz Company in Pittsburgh, Pa.

DR. SHORE received his M.D. degree from Temple University School of Medicine in 1946 and served his internship at Allegheny General Hospital in Pittsburgh. He is a member of the American Medical Association and the American Academy of General Practice.

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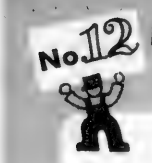



**in a series of washroom survey comments from "SBS soap counselors"**

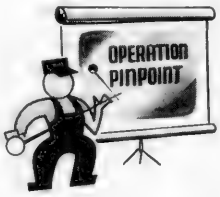
*We showed them how to remove tough soils the easy way!*

**Waring G. Houston**

SBS Soap Counselor, reports how he aided a rubber manufacturing plant in solving a specific soil problem:

"This company has always had a tough time keeping its washrooms clean because of the presence of rubber dust, latex, cements, special inks and other hard-to-remove soils. Maintenance men resorted to special-purpose cleaners to cope with the problem. In a short time, the cost of labor and cleaning materials went sky high!

"We pointed out, of course, how much easier they could do the total cleaning job with SBS-50 Cleaner-Disinfectant-Sanitizer-Deodorant. We demonstrated SBS-50's effectiveness against every tough soil found in their washrooms. They were amazed to find out how easily they could end the costly duplication of manpower and materials!"

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## Calendar of Meetings

December 6, 1961—New York State Society of Industrial Medicine annual meeting, New York University Club, New York City. Executive Secretary: R. MacDiarmid, M.D., Room 2400, 30 Rockefeller Plaza, New York 20, N. Y.

December 9, 1961—Central States Society of Industrial Medicine and Surgery, Knickerbocker Hotel, Chicago, Ill. Secretary: A. P. Skoog, M.D., 55 East Washington St., Chicago 2, Ill.

February 7-9, 1962—American Academy of Occupational Medicine, Pittsburgh-Hilton Hotel, Pittsburgh, Pa. Secretary: Paul J. Whitaker, M.D., Allis-Chalmers Mfg. Co., P.O. Box 512, Milwaukee 1, Wis.

February 22, 1962—Industrial Medical Association of Upstate New York, Towne House Motor Inn, Rochester, N. Y. Secretary: Dallas E. Billman, M.D., Corning Glass Works, Corning, N. Y.

March 30, 1962—Michigan Industrial Medical Association, Parke, Davis & Company Research Laboratories, Ann Arbor, Mich. Secretary: Robert D. McIntosh, M.D., 6307 West Fort St., Detroit, Mich.

**April 10-12, 1962—Industrial Medical Association, Pick-Congress Hotel, Chicago, Ill.** Managing Director: Clark D. Bridges, 55 East Washington St., Chicago 2, Ill.

May 6, 1962—Carolina Industrial Medical Association, Raleigh, N. C. Secretary: B. W. Goodman, M.D., 12 Second Ave., N.E., Hickory, N.C.

May 13-17, 1962—American Industrial Hygiene Association, Sheraton-Park Hotel, Washington, D.C., Executive Secretary: George D. Clayton, 14125 Prevost St., Detroit 27, Mich.

October 2-3, 1962—Congress on Occupational Health, Somerset Hotel, Boston. B. Dixon Holland, M.D., Director, Department of Occupational Health, American Medical Association, 535 North Dearborn, Chicago 10, Ill.

October 12-13, 1962—Western Industrial Health Conference, Jack Tar Hotel, San Francisco, Calif. Secretary: Birchard M. Brundage, M.D., Rocketdyne, 6633 Canoga Ave., Canoga Park, Calif.

October 15-19, 1962—American Public Health Association, Hotel Fountainebleau, Miami Beach, Fla. Executive Director: Dr. Berwyn F. Mattison, 1790 Broadway, New York 19, N. Y.

October 24-25, 1962—Industrial Hygiene Foundation, Mellon Institute, Pittsburgh, Pa. Managing Director: Dr. H. H. Schrenk, 4400 Fifth Ave., Pittsburgh 13, Pa.

Severe crushing injury with degloving of entire hand. Despite marked vascular deficiency, it was hoped that the thumb and index finger could be saved. Residual skin flaps were closed and raw areas covered with Furacin impregnated gauze.

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Utilizing the principle of delayed closure, major defects were covered in several stages with skin grafts and an abdominal flap. Index finger was later reconstructed to improve function. Final result shows useful, opposing thumb and index finger.

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# A REPORT ON DIET MANUALS AND PHYSICIANS' DIET PADS

A dietitian consultant from a leading metropolitan hospital examined a report covering 66 hospital diet manuals. Breakfast cereals were included in all general diet plans. Another professional consultant found in a study of 60 physicians' diet pads that a cereal breakfast was included in 44 of them. All low-fat diets in these pads included a moderate low-fat, well-balanced cereal breakfast. This demonstrates that cereal is a basic breakfast food, because it is included in the above nutrition materials.

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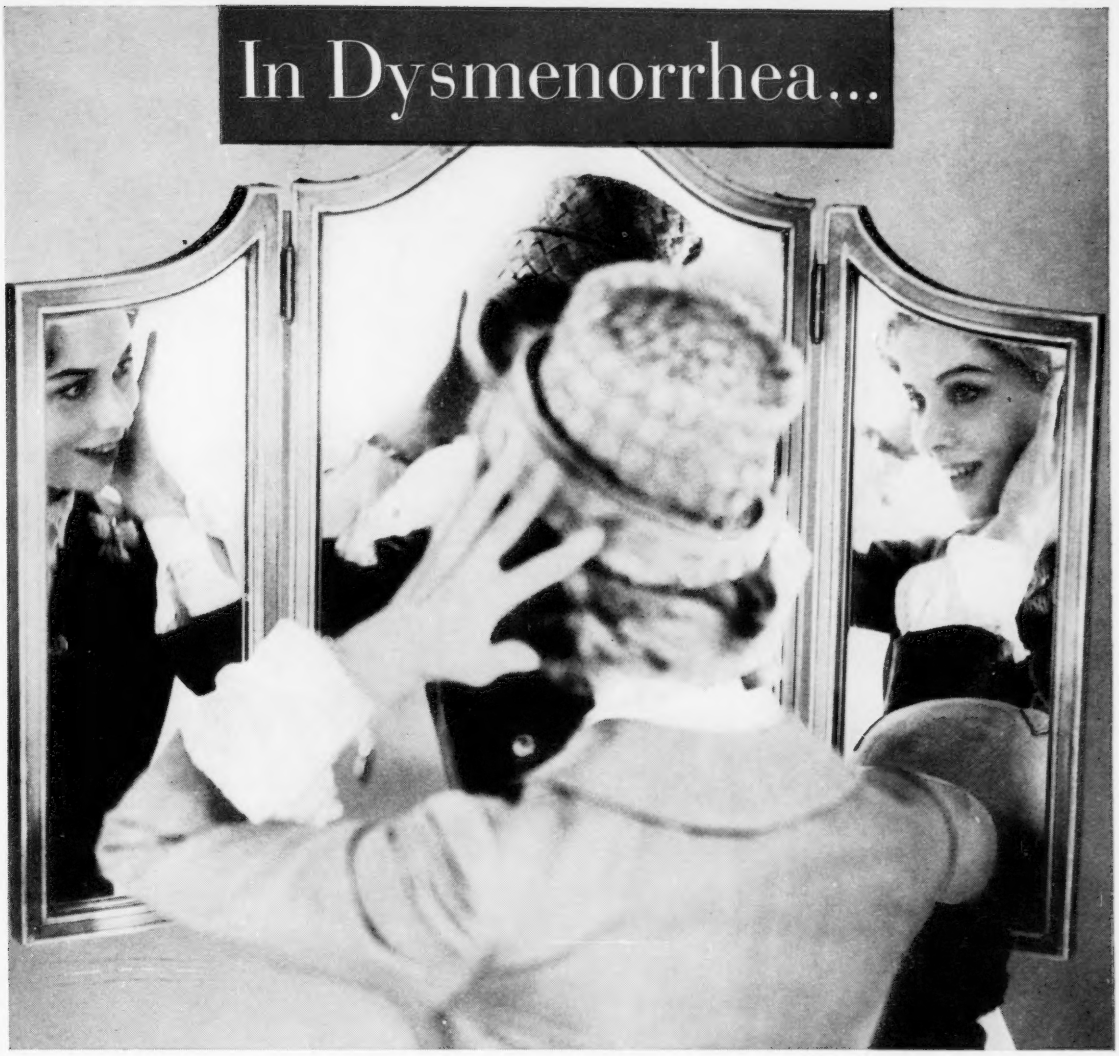


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Aspirin.....(5 grains) 300 mg.
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**Trancoprin is more than a simple analgesic:** It deals with cramping pains in three ways. Besides dimming pain perception, Trancoprin, through its tranquilizing action, reduces anxiety and raises the tolerance for discomfort. And, against the spasm caused by pain which, in turn, produces more pain, Trancoprin exerts its skeletal muscle relaxant action.

**Trancoprin is exceptionally safe to use:** Fewer than two and a half per cent of patients can be expected to have any side effects, and these are of a minor nature.

Available in bottles of 100 tablets. The usual dosage in dysmenorrhea is 2 tablets 3 or 4 times daily.

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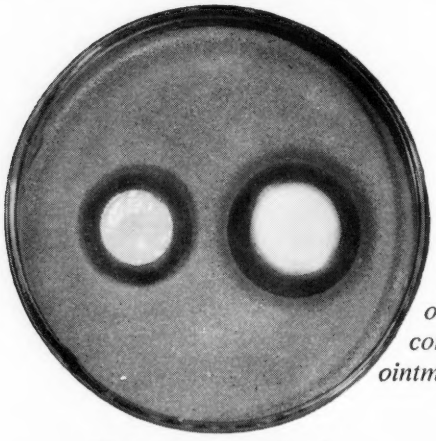
Before prescribing be sure to consult Winthrop's literature for additional information about dosage, possible side effects and contraindications. 1602M

# *If 2 ointments contain the same antibiotics, how can one be more effective?*

...The unique base* of Neo-Polycin releases a greater concentration of antibiotic into the lesions.



*Neo-Polycin Ointment on right conventional grease-base ointment on left*

**Agar incubations show Neo-Polycin® Ointment more effective against common topical pathogens**

This agar plate, containing *Staph. aureus*, was incubated for 24 hours at room temperature. Note the greater zone of inhibition around Neo-Polycin Ointment (right), than around the grease-base ointment of comparable antibiotic content (left). Tests on the following pathogens gave similar results: *beta hemolytic strep., E. coli, Proteus vulgaris, Pseudomonas aeruginosa.* Higher concentrations of antibiotics released from Neo-Polycin tend to inhibit the growth of relatively resistant strains and minimize antibacterial resistance created by sub-effective concentrations.

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